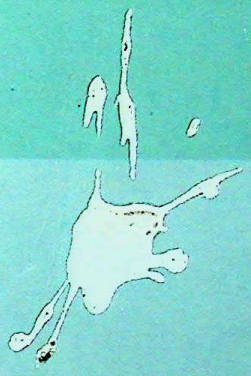
Gandhi Memorial College Of Education Bantalab Jammu

MKR Krishnan

Handbook of Forensic Medicine and Toxicology



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V V Pillay

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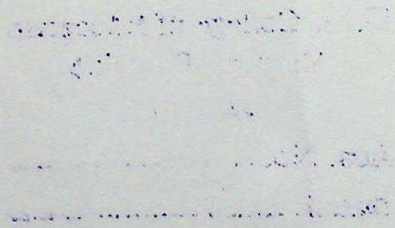

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MKR Krishnan

Handbook of Forensic Medicine and Toxicology

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This edition is dedicated to some of the eminent Forensic Experts of Kerala, a state which has consistently maintained an excellent standard of medicolegal work ever since its inception:

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Handbook of Forensic Medicine and Toxicology

12th Edition 2001

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Credits for 12th Edition

VVP	▪ Chapters 3, 5, and 21 (FM) ▪ Chapters 1 to 14 (Tox) ▪ All Boxed Items, Annexures, Case Histories, Quotations ▪ Indexing ▪ Over-all Editing & Proof reading
JTJ	▪ Chapters 8, 9, 10, and 12 (FM)
VRV	▪ Chapters 11, 13, 18, and 19 (FM)
WV	▪ Chapters 16, 17, and 20 (FM)
SB	▪ Chapters 4, 14, and 22 (FM)
AK	▪ Chapters 1, 2 and 15, along with Case Histories (FM)
KMS	▪ Chapter 7 (FM)
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SS	▪ Chapter 23, along with Case Histories (FM)

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Foreword

'Knowledge' or 'Wisdom' gets assimilated if it is poured into deserving hands. So the meticulous and untiring efforts of Prof. Pillay in bringing out the newly edited "Handbook of Forensic Medicine" is definitely most rewarding.

'Adequate Trained Personnel' and 'Relevant Literature' are essential cornerstones of any discipline. Forensic medicine too, is no exception. The Ministry of Home Affairs of Government of India once remarked, "The status of medico-legal practice throughout the country is in a deplorable condition". It is true that inadequacy of acumen can lead to erroneous conclusions, which in turn can affect the quality of the profession, and the effective discharge of duties to the common man.

Medico-legal practice in India has often taken the brunt of hardcore criticism. The pioneers in the field of forensic medicine have done a lot for the betterment of the discipline, and today many standard textbooks are available to teach and guide young forensic professionals. Still, there remains much more to attain – the subject needs further upgrading and updating; it needs to synchronise with Indian requirements; it needs renovation to satisfy the challenging demands of forensic scientists.

Prof. Pillay's earnest effort to edit 'Dr. Krishnan's Handbook of Forensic Medicine' is most laudable. He places the subject in the light of the present day demands, and has produced a book which would be a great asset to the experts, and to budding scientists of this vital medical discipline. His efforts to revitalize the book with case histories and illustrations have resulted in giving new life and style to the whole subject. The revision and updating that he has made, will definitely appeal to every reader. The list of references incorporated in every chapter lends the book authenticity and credibility. The renovations that are made to toxicology section need special mention.

Through this praise-worthy contribution, Prof. Pillay has attained a coveted position among the stalwarts of forensic medicine. I am sure that the book in its new shape will be a standard text book of forensic medicine for the graduate and postgraduate trainees, a guiding light in the hands of the faculty and practising doctors, and a useful tool for the enforcement of medical jurisprudence. It is my sincere belief that Universities in India and abroad will come forward to prescribe it as a standard textbook of forensic medicine and toxicology.

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Preface to 12th Edition

This *Handbook of Forensic Medicine and Toxicology* has been around since 1965, and has seen eleven editions and numerous reprints. It should have become a classic treatise on the subject by now due to constant revision and improvement. And yet, sadly, the full potential has somehow never been realised. The reasons are not difficult to surmise, (hard though it may be to face them). To put it plainly, even bluntly, the main reason has been just a lack of genuine effort on the part of previous authors/editors in updating the subject, and making it practically useful to medical students as well as doctors dealing with medicolegal work.

It has always been a source of amazement to me that a subject as inherently fascinating as Forensic Medicine, can be presented in a dull and uninteresting manner as is the case with some of the existing books on the subject. To a certain extent, one could say Toxicology is tedious or monotonous, but even here, much can be done to enliven its presentation. All it takes is a sincere effort at stimulating the interest of the readers by way of clear description, lucid language, stress on fundamental facts, and illustrative case histories. A judicious mix of such a masala cannot but fail to kindle the hunger for acquisition of knowledge in every student!

It was with this firm conviction that I embarked upon the challenging task of editing this book (nay, revamping it), with help from some very competent colleagues. Each one of these Sub-Editors was given certain specified topics to update, along with explicit guidelines as to the manner of presentation. What you have in your hand now is a completely rejuvenated handbook of Forensic Medicine and Toxicology that bears little resemblance to its previous incarnations! We of the Editorial team sincerely hope that the book will be considered as a refreshing departure from conventional handbooks on the subject, in being both practically useful as well as eminently readable. While the entire team is collectively responsible for the plus points of this book, I should be held solely responsible for its drawbacks and deficiencies.

So, if you get to like this book do tell the world about it, but if you don't, just tell me! Suggestions for improvement would be greatly appreciated.

Dr. V V PILLAY
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SECTION - A

Forensic Medicine

"The aim of a Forensic Expert dealing with criminal cases should be objectivity, not neutrality".

- Peter Vanezis

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CHAPTER - 1

Legal Procedure

"The net of law is spread so wide, no sinner from its sweep may hide".

- J. J. Roche

FORENSIC MEDICINE

Forensic medicine is a branch of medicine which deals with the application of medical knowledge in the administration of law and justice. It has also been defined as *the science which applies the principles and practice of medicine to the elucidation of questions in judicial proceedings.*[1] A medical practitioner may have to on occasion, give evidence as an expert witness in a court of law to prove the innocence or guilt of an accused, or to authenticate or disprove a criminal charge of assault, rape, or murder brought against an individual.[2]

Forensic Medicine is a vast subject, and requires a thorough knowledge of all branches of medicine. The medico-legal expert needs to take the help of various subjects to arrive at conclusions. He should be aware of the regulations, statutes, and acts relating to the practice and obligations of medical practice. A knowledge of legal procedure and some sections of the **Indian Penal Code** (I.P.C), **Criminal Procedure Code** (Cr.P.C), and **Indian Evidence Act** (I.E.A), related to various offences, is also desirable.

In the ensuing sections and chapters of this book, an attempt has been made to present the subject matter relating to Forensic Medicine and Toxicology in accordance with the revised guidelines for undergraduate medical curriculum as suggested by the Medical Council of India.[3]

LEGAL PROCEDURE

Whenever a person dies, the cause of death has to be determined. If the death is due to natural causes like cancer, pneumonia, heart failure, etc., no further investigation is necessary and the body can be handed over to the relatives for burial or cremation, or any kind of disposal as per their religious beliefs. If there is any suspicion of foul play, or if the mode of death seems to be unnatural, it must be reported to the authorities concerned. An immediate investigation (**inquest**) has to be conducted.

INQUEST

An inquest is a legal inquiry into the circumstances and cause of death, in cases of sudden, suspicious, or unnatural deaths.

Types of inquest:

1. Police Inquest
2. Magistrate's Inquest
3. Coroner's Inquest
4. Medical Examiner's system.

Police Inquest

This type of inquest is conducted by an officer in charge of a police station. On receipt of informa-

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tion of an unnatural death, the police officer informs the nearest Magistrate, and proceeds to the place of death. He then conducts an inquest in the presence of two or more respectable inhabitants of the neighbourhood (**panchas**).

After the necessary investigation, a report is drawn up on the apparent cause of death as judged by the state of the body, the injuries present on the body, hearsay evidence, circumstantial evidence, etc. This document is called **panchanama** or **inquest report**. This is signed by the police officer and the persons present at the inquest. If any foul play or unnatural death is suspected, the police officer forwards the body for postmortem examination to the nearest medical officer, along with written request and a copy of the panchanama, to help him find out the probable cause of death. If the autopsy report confirms that the death is due to an unnatural cause, further enquiry and trial of the case is carried out in the usual manner by the magistrate concerned, to whom the entire records of the case are transferred by the police officer.

Coroner's Inquest

Coroner's inquest is a type of inquest conducted by a **Coroner** who is either a medical professional with legal qualifications, or a graduate in law. He is governed by the **Coroner's Act (1871)**, which prescribes his powers, duties, and conduct. He holds an inquest on receipt of information of any of the following:

1. Sudden death
2. Murder, infanticide, or suicide
3. Unnatural death
4. Unexplained or obscure death
5. Death by accident or poisoning
6. Death during a surgical operation
7. Death of a convict in jail
8. Death during surgery
9. Death of a person in police custody
10. Death of a patient in a mental hospital

The Coroner's Court is a court of enquiry, and not of trial. He is empowered to summon witnesses and record their evidence on oath. He can order for postmortem examination and exhumation whenever required.

Until recently, the Coroner's Inquest existed in the city of Mumbai, from where it has now been abolished. Previously it was also conducted in various other cities such as Calcutta. Today this system of inquest does not exist anywhere in India. It is however the prevalent method of inquiry in the U.K and some states of the USA.

Magistrate's Inquest

A magistrate conducts inquest in the following circumstances :

1. Dowry death
2. Death of a convict in prison
3. Death of a person in police custody
4. Death of a person as a result of police firing
5. Death in mental hospital.
6. Exhumation

Medical Examiner's System

Medical Examiner's System is a type of inquest which is prevalent in most of the states of USA.

All sudden, unexpected, or unnatural deaths are analysed by a Medical Examiner who is a forensic pathologist. This type of inquest is considered superior to all other types of inquest, because it is conducted by a forensic pathologist with medical, scientific, and legal knowledge.

Several years ago, a group of experienced forensic pathologists who were members of the Pathology-Biology section of the American Academy of Forensic Science, created the National Association of Medical Examiners (NAME)[4]. It functions as a resource for management studies relating to

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medical examiners practice. NAME has several members who, through experience and/or special study, possess organisational study skills suitable for regulating the functioning of the Medical Examiner's system.

COURTS

Members of the medical profession may be required to attend court to give evidence in medico-legal cases. Therefore it is desirable that all medical practitioners possess some basic knowledge about criminal courts and court procedures.

Criminal Courts of India

Table 1.1 gives the names, locations, and powers of the criminal courts in India.

Special Magistrates: are generally appointed for special purposes, when the other regular magistrates cannot cope with a sudden and unexpected extra load of work, or when the enquiry has to be completed within a stipulated time. Special Magistrates maybe of any class.

Table 1.1 Criminal Courts of India.

S. No.	Court	Location	Powers
1.	Supreme Court	New Delhi	Highest judicial tribunal of the country. Can pass any sentence. Usually considers appeals from lower courts.
2.	High Court	Capital of every state (usually)	Highest judicial tribunal of the state. Can try any offence and pass any sentence.
3.	Sessions Court (District Sessions Court)	District Headquarters	Highest judicial tribunal of the district. Can try only those offences which have been committed to it by a magistrate. Can pass any sentence, but death sentence must be confirmed by High Court.
4.	Magistrate's Courts are of two types: I. *Judicial Magistrate* a. i. Chief Metropolitan Magistrate ii. Chief Judicial Magistrate	Metropolitan area District (not being a metropolitan area)	Can pass a sentence of imprisonment upto seven years; fine without limit.
	b. Judicial Magistrate of I class Metropolitan Magistrate	Subdivision of district Metropolitan area	Can pass a sentence of imprisonment upto three years; fine upto Rs. 5,000/-
	c. Judicial Magistrate of II class		Can pass a sentence of imprisonment upto 1 year and fine upto Rs. 1,000/-
	II. *Executive Magistrate*	I/c District, subdivision, or Taluk etc.	Appointed by the State Government; usually officers of Revenue dept., District Collector, Sub Collector or Tahasildar.

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Juvenile Magistrates: preside over juvenile courts and they try juvenile offences, i.e., those committed by individuals below the age of sixteen years(male), or eighteen years (female). Juvenile Magistrates are generally women.

PROCEDURE IN COURT

Subpoena or **Summons** is a document commanding the attendance of a witness in a court of law, under a penalty (*sub*-under, *poena*-penalty). If the summoned witness fails to attend without genuine reason, he can be arrested and produced in court. A summons is issued by the court, in writing, signed by the presiding officer, and is served on the witness by a police officer or an officer of the court, or any other person specifically authorised for the purpose. It is issued in duplicate, one of which is to be returned after signing, while the other is retained by the witness. A summons must be implicitly obeyed, unless there are valid and urgent reasons. A witness who wilfully avoids to appear before a court after having been duly served a summons, is guilty of contempt of court. The penalty for non-compliance of summons is a warrant of arrest and a compulsory appearance before the court. In the matter of attending a court in response to a summons, it must be borne in mind that a witness must give priority to a criminal case over a civil case, if he is summoned to two courts at the same time, because criminal cases have priority over civil matters. Similarly, if he is summoned by two criminal courts on the same day, he is to give preference to the higher court which takes precedence over a lower court.

Conduct Money: In civil cases, the medical officer is usually paid money to meet his travelling expenses from his place of residence-to the court, and back. This is called **conduct money**. This is paid to him at the time the summons is served on him. If this amount is not paid to him, he may ignore the summons, if he so desires. If he feels the sum is inadequate, he can inform the court accordingly and get it enhanced. In criminal cases however, no such fee is given usually, and it is the bounden duty of every citizen to attend the court whenever summoned.

Oath

Let no man swear on oath falsely even in a trifling matter, for he who swears on oath falsely, is lost in this world and ever after .

- Manu Smrithi

Once the Medical Officer who is summoned makes his appearance in court, his evidence is recorded after administration of the oath. An **oath** is a declaration required by the law, to be made before the court by the witnesses, to speak the truth, the whole truth, and nothing but the truth. The oath is administered to the witness by the court clerk.

Once a witness has been sworn, he is liable to be prosecuted for **perjury**, (wilful utterance of falsehood) if he does not speak the truth (punishable under sec 193 IPC).

After having been sworn, the evidence of the witness is recorded in the following manner:

1. Examination -in-chief
2. Cross examination
3. Re-examination
4. Court questions

Examination-in-Chief: Examination -in-chief is the examination of the witness by the counsel (advocate) of the party which has called him. In government prosecution cases, the public prosecutor commences the examination-in-chief, the purpose of which is to appraise the court of all the facts of the case. Leading questions are not allowed in examination-in -chief, except when a hostile witness is being examined. A **leading question** is one which itself suggests the answer. For e.g., "*Doctor, was not the injury caused by a sharp weapon*?", is a

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leading question. Normally, the question should be phrased in this manner - "*Doctor, by what weapon has this injury been caused ?*".

Cross Examination: Cross examination follows examination-in-chief. It is the examination of the witness by the counsel (advocate) of the opposite party, namely counsel for the accused. The purpose of cross examination is to highlight inaccuracies and contradictions in the statements of the witness, and thereby to prove to the court that this evidence is not valid. **Leading questions** are allowed during cross examination. The witness must therefore be very careful in answering the questions. He may be asked questions to test his truthfulness, skill, and character. A medical witness must not feel insulted, must avoid losing his temper, and must answer the questions with dignity and poise.

Re-Examination: Cross examination is followed by re-examination. It is conducted by the counsel who has conducted the examination-in-chief. The purpose of re-examination is to clear any doubts that may have arisen during cross examination. Leading questions are not allowed. No new matter may be introduced without the permission of the judge and the consent of the counsel of the opposing party. In the event of a new topic being introduced, the opposing party has the right to cross examine on the new matter.

Court Questions: These are questions put forward by the presiding officer of the court. These questions are posed to clear doubts, if any.

MEDICAL EVIDENCE

The term "*Evidence*" means all information which the court permits, which is helpful in relation to matters of fact under enquiry. It may be **oral** or **documentary**. Evidence stated orally is called **oral evidence**, and that which is produced in the form of documents is called **documentary evidence**.

DOCUMENTARY EVIDENCE: This comprises documents produced before the court, and includes:

1. Medical certificate
2. Medico-legal report
3. Dying declaration.

Medical certificates: These are certificates which pertain to sickness, insanity, death, etc. They are issued only by a registered medical practitioner and are admissible in court. It is however, an offence to issue a false certificate. When a medical practitioner is not sure of the cause of death or when he suspects foul play, he must refuse to issue a death certificate and must inform the police.

The Registration of Births and Deaths under Municipal Acts stipulates that notice must compulsorily be given by a doctor in attendance, at the time of the birth or death of an individual[5]. Hence it becomes mandatory for a medical practitioner to issue a death certificate after inspecting the body and satisfying himself of the person's death, stating the cause of death.

The medical practitioner is not entitled to charge any fee for issuing a death certificate. He should not refuse nor cause unnecessary delay in issuing a death certificate. The recommended format of death certificate is mentioned below.

Form of Medical Certificate of Cause of Death

The format of the certificate proper (medical part) conforms to the standard prescribed by the WHO, and has the following features:

Cause of Death	Interval between onset & death approx:
I.	
1. ***Immediate Cause:*** (State the disease, injury or complication, which caused death, not the mode of dying such as heart failure, asthenia etc.) a)	

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2. ***Antecedent Cause***:
(Morbid conditions if any, giving rise to the above cause, stating the underlying conditions last)
b) .....

II. Other significant conditions contributing to the death, but not related to the disease or conditions causing it.
.....
.....

Medico-legal Report

Medico-legal reports are documents prepared by medical officers in compliance with a written requisition issued by a magistrate or police officer. Medico-legal reports may pertain to injury, sexual offence, death, etc. It contains all the facts observed by the medical officer, and his opinion drawn therefrom.

His opinion must be based upon the observations made by him, and not on hearsay evidence.

Dying Declaration

A dying declaration is a statement, written or oral, made by a person who is on the verge of dying as a result of unnatural causes. The statement should relate to the cause of his imminent death, or any of the circumstances of the transaction resulting in his present condition.. Such a declaration is admissible in court, when the cause of that person's death is the subject of enquiry. It may provide useful information and help to obtain justice. A person who is accused of a crime cannot make a dying declaration, but only a person who is the victim of a crime. Statements unconnected with the actual crime are not admissible in a dying declaration.

It is believed that a person who is about to die will speak only the truth. A dying declaration has the same measure of worth, as that of a statement made on oath, in the court. A dying declaration is valid even if the declarant lives for some days after making the declaration, but in that case, it may be relied on only as corroborative evidence.

As soon as a doctor knows that the victim of a crime who is in danger of dying, has something of importance to say to the court, he must send for a magistrate. If the doctor feels that the declarant may die before the arrival of the magistrate, he can record the declaration himself. It is essential to certify that the declarant was in a state of sound mind (*compos mentis*) at the time of making the declaration.

Although no oath is administered, a dying declaration is admissible in court as if this had been done, provided it is recorded as per the guidelines laid down for the purpose. It must be recorded in the presence of two witnesses. It is to be recorded verbatim, in the language in which the victim prefers to speak. No information must be elicited by asking leading questions. No prompting or suggestions should be made to the declarant, and undue influence must not be imposed on the declarant.

The investigating police officer should not be present when the declaration is being made. If the dying person is unable to speak, but is able to make gestures in answer to questions, it can be recorded after deciphering the gestures. The medical officer recording the declaration should only mention the precise nature of the gestures so made, and not interpret them himself. Interpretation is done subsequently by the court.

After the complete declaration has been recorded, it must be read over to the declarant, and must if possible, be signed by him. It must then be attested by the writer and the witnesses. If the declarant dies or becomes unconscious while the declaration is being made, the medical officer writing it must record as much information as he has obtained and sign it. It must then be forwarded to the magistrate in a sealed envelope.

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If a dying declaration is recorded by a magistrate, and in the presence of the accused who is given an opportunity to cross examine the declarant, it is called a **dying deposition**. This is however not prevalent in India. A dying deposition has greater value than dying declaration in court, for the reason that it is recorded by a Magistrate, in the presence of the accused who is permitted to cross examine the declarant.

ORAL EVIDENCE: Oral evidence refers to evidence which is tendered verbally. Oral evidence, as far as possible, must be direct, i.e, if it refers to a fact which can be seen, heard or perceived, then it must be the evidence of a person who says he saw, heard, or perceived it. In other words, it must be the evidence of the actual witness. However, it is not always necessary that oral evidence must be direct. Exceptions to direct oral evidence include:

a) dying declaration
b) expert opinion expressed in a treatise, when the author is dead, or cannot be found, or is otherwise incapable of giving evidence
c) deposition of medical witness in a lower court
d) chemical examiner's report.

Hearsay evidence: Hearsay evidence is the evidence of one who has no personal knowledge of the facts of the case, but just repeats what he has heard others say. For e.g., if Mr. A has seen Mr. B shooting Mr. X with a revolver, and if Mr. A testifies that he has witnessed this incident, then it is **direct evidence**. But if Mr. A has got an information from Mr. Z that Mr. B shot Mr. X, then it is **hearsay evidence**, and it is not permissible in court, as the witness cannot be cross examined.

Oral evidence is superior to documentary evidence, because it can be subjected to cross examination.

CIRCUMSTANTIAL EVIDENCE is the evidence obtained from suspicious circumstances. For e.g., the sudden disappearance of a person who is suspected to be the criminal. In some cases, circumstantial evidence is more reliable than eye witness accounts.

WITNESSES: Witnesses are of two kinds, namely, **common witness** and **expert witness**. A common or ordinary witness, is one who testifies only to the facts observed by him. His evidence requires only common intelligence and knowledge. He is not capable of forming opinion or drawing conclusions from the facts observed by him.

An expert witness on the other hand, is one who is skilled in scientific, technical, or professional matters, and who on account of his professional training, experience, and ability, is capable of forming opinions, or drawing inferences.

Sometimes a witness deliberately conceals the truth or gives false evidence. After making a particular statement in court, he contradicts this subsequently. He is therefore presumed to have an interest or motive for concealing the truth, or giving false evidence. The court therefore declares him to be **hostile**. It is possible that a common witness or expert witness may turn hostile. When a hostile witness is being examined, leading questions are permissible even during examination-in-chief.

CONDUCT OF A DOCTOR IN THE COURT[6]

1. Go to court well prepared with the details of the case. Take care to have answers ready for anticipated questions.
2. Be in possession of all relevant certificates, documents, and notes.
3. Do not attempt to memorise anything, since you are allowed to refresh your memory by consulting your report or notes.
4. Be well dressed and modest.
5. Speak audibly and clearly.
6. Use simple language, and avoid technical terms as far as possible.
7. Do not exaggerate anything. Avoid the use of superlatives, e.g., "It was the *biggest* wound I have ever seen.....".
8. Do not fumble while referring to any case notes or records.

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9. Do not discuss the case with anybody except the lawyer who has called you.
10. Address the Judge respectfully - "*Sir*" or "*Your Honour*". When entering the room, bow to him before taking your seat.
11. Avoid discrepancies between your earlier statements and your testimony to the court.
12. Do not evade any question. If a question is unclear, ask for it to be repeated or rephrased.
13. If you do not know the answer to a particular question, be frank and admit it.
14. Do not lose your temper. Always remain calm, composed, and courteous .
15. Do not argue. Disagree firmly with anything you are not convinced about. But do not give the impression of being dogmatic.
16. Do not give rambling, long winded answers. Try to be brief and precise.
17. As far as possible do not volunteer any statement on your own. However if you feel that by not making some statement, injustice may result, do mention it.
18. Express opinions only on the basis of your own knowledge and experience. You can however utilise published opinion on scientific facts by qualified writers.
19. Be honest. If you have made some mistake, admit it. Do not attempt to cover up any lapses or errors.
20. Be absolutely impartial. Even though you may have been called by the prosecutor, present your facts objectively without taking sides. Remember the following quotation made by **Brouardel**, an eminent French medico-legal expert :

"If the law has made you a witness, remain a man of science. You have no victim to avenge, no guilty man to convict, and no innocent man to save. You must bear testimony within the limits of science."

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CHAPTER - 2

Medical Law and Ethics

"Ignorance of the law excuses no man. Not that all men know the law, but because it is an excuse every man can plead, and no one can refute him".

- John Selden

MEDICAL JURISPRUDENCE

('*Juris*' = law, and '*Prudence*' = knowledge)

Medical jurisprudence is the study of law in relation to medicine. In other words, *it deals with the legal aspects of medicine such as the legal rights, privileges, duties, and obligations of a medical practitioner.* Medical practitioners must be aware of legal implications on their medical practice. They should be familiar with the various statutes, rules and regulations that are in force, and which have been enacted by the Central or State government, as well as possess a working knowledge of the functioning of statutory bodies and Medical Councils. If a medical practitioner is not familiar with the relevant laws and regulations, he may find himself inadvertently contravening these, and thereby court trouble.

INDIAN MEDICAL COUNCIL

The Indian Medical Council (IMC) is a body that is intimately concerned with the medical profession, and is governed by the **Indian Medical Council Act (1956).** It consists of a number of members elected from various states and universities of India, and some members nominated by the Government of India. These members hold office for a period of 5 years. They elect a President and a Vice-President from among themselves. The council also appoints a Registrar and a Secretary for day to day functioning.

FUNCTIONS OF THE INDIAN MEDICAL COUNCIL

a) **Medical Register**
The Indian Medical Council (IMC) maintains a register. The register contains the names, addresses, and qualifications of all medical practitioners who have registered themselves with any state medical council. When a medical graduate with a recognised medical qualification registers himself with any State Medical Council (SMC) after paying the prescribed fee, notification is sent to the IMC and his name gets registered in the Indian Medical Register also. Similarly, if the name of a medical practitioner is erased from the State Medical Register for any reason, it is automatically deleted from the Indian Medical Register also.

b) **Medical Education**
The IMC regulates the standards of medical education. It appoints a committee for both undergraduate and postgraduate education. The respective committee appoints inspectors to visit various institutions. The purpose of such a visit is to assess the facilities provided by the institutions, and to find out whether they are on par with the specifications prescribed by the IMC.

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Inspectors are also required to visit these institutions during examinations, in order to assess the standard of such examinations.

On the basis of the report made by the inspectors, the council recommends to the Government of India, due recognition or derecognition of a particular institution. Often, degrees are derecognised only temporarily, and guidelines are given on ways and means of improving the standards in order to obtain recognition subsequently.

The recommendation of the council is mandatory for the Central Government to act, either for recognising or derecognising an institute or a degree awarded by an institute.

c) **Recognition of Foreign Medical Qualifications**
The IMC maintains three schedules.

The **First schedule** contains a list of medical degrees offered by different universities in India, which are recognised by the council.

The **Second schedule** contains a list of medical degrees offered by different universities outside India, which are recognised by the council.

The **Third schedule** has two parts.
Part A contains a list of medical qualifications conferred by Indian universities or institutions which are not mentioned in the first schedule.
Part B contains a list of standard medical qualifications of foreign countries, not mentioned in the second schedule. These degrees may be recognised when Indian citizens obtain them and apply to the council individually for recognition.

d) **Apellate Tribunal**
The IMC acts as an appellate tribunal. If any registered medical practitioner who has been penalised by any SMC desires to appeal against such punishment, he can make a representation to the Central Government, which will subsequently consult the IMC. The IMC will examine the facts of the case, and then furnish its recommendations to the Central Government, which will be binding on the appealing party as well as the SMC concerned.

e) **Disciplinary Control**
The IMC exercises disciplinary control over members of the medical profession. The council prescribes minimum standards of professional conduct, ethics, and etiquette. It issues a **warning notice** periodically which is a list of offences constituting infamous conduct.

STATE MEDICAL COUNCIL

Every state in India has a State Medical Council (SMC), located usually in the capital of that state. It is composed of members of the medical profession elected from among themselves, as well as those nominated by the State Government. A President and a Vice President are elected by the members from amongst themselves.

FUNCTIONS OF THE STATE MEDICAL COUNCIL

a) **Medical Register**
The SMC maintains a register containing the names, addresses, and qualifications of those medical practitioners who have registered with the council. It also contains the date on which a particular degree was obtained, and the name of the university which conferred it. This register is maintained by a Registrar who is appointed by the council.

On receiving further qualifications, the medical practitioner concerned must inform the Registrar of the same.

The Registrar is expected to inform the IMC without delay, all the additions and amendments

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made from time to time in the State Medical Register.

b) **Disciplinary Control**
The council exercises disciplinary control over registered medical practitioners, and has the power to delete the names of those found guilty of professional misconduct. Such deletion may be temporary or permanent. If an appeal is made by an aggrieved medical practitioner against the punishment awarded, and if the IMC makes a recommendation in favour of the appellant, his name will be restored in the State Medical Register.

c) **Warning Notice**
The council makes clear disclosure of those practices it considers as constituting infamous conduct. It issues warning notices to the registered medical practitioners found indulging in unethical practice, and advises them to conduct themselves according to the ethical norms prescribed by the council.

PROCEDURE OF DISCIPLINARY ACTION

The State Medical Council initiates disciplinary action in any of the following situations:

1. When a complaint is made by a person or persons against a registered medical practitioner.
2. When a court of law convicts any registered medical practitioner for an offence involving moral turpitude.

The complaint is received by the Registrar. He submits the complaint to the President who refers it to a subcommittee or an executive committee.

The committee examines the complaint and decides whether it is necessary to take action against the medical practitioner concerned. If there is *prima facie* evidence of misdemeanor, a notice is issued to the medical practitioner to be present on a specified day to face the charges. The defendant medical practitioner and his legal advisor should be present on the appointed day.

First, the charges are read out with relevant details. The counsel for the complainant outlines the nature and substance of allegations with relevant evidentiary material. Then the defendant (registered practitioner) or his legal advisor rebutts the charges. After hearing both sides, the members of the council are asked to express their opinion. The judgement is based on the majority view.

If the council finds *prima facie* evidence, and a majority of the members feel that the offence is proved, the guilty registered medical practitioner is punished in one of the following ways:

I. **Warning**: A warning is issued to the medical practitioner to conduct himself according to the expectations of ethical standards prescribed by the council.
II. **Temporary erasure**: The name of the medical practitioner is erased from the register for a specified period after which it is restored.
III. **Penal erasure**: The name is permanently removed from the register. This is also known as *professional death sentence*.

RIGHTS AND PRIVILEGES OF A REGISTERED MEDICAL PRACTITIONER

There are certain rights and privileges that accrue to a medical practitioner as a result of registration in a medical council, and they are as follows :

a) Right to practice the profession anywhere in India
b) Right to add professional titles to his name.
c) Right to choose patients.
d) Right to prescribe or dispense medicines
e) Right to perform a surgical operation.
f) Right to seek government employment as a doctor.

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g) Right to issue birth, sickness, insanity and other certificates which will be held valid in a court of law.
h) Right to give evidence as an expert witness in a court of law.
i) Right to possess, dispense, or prescribe drugs listed in the Dangerous Drugs Act.
j) Right to claim payment of fees for professional services rendered.

RED CROSS EMBLEM

The objectives of the Red Cross Society are closely allied with those of the medical profession. This has led to the mistaken belief on the part of some medical practitioners, that they are entitled to use the Red Cross emblem[1].

This belief is erroneous, and in fact the law prohibits its misuse by medical practitioners. The right to display the Red Cross emblem (a red cross on a white background) can be exercised only by the members of the medical division of the armed forces of a country.

As per the **Geneva Convention Act 1960** (to which India is a signatory), *Sec. 12* prohibits the use of the Red Cross emblem and all other allied emblems for any purpose whatsoever, without approval of the Government of India, while *Sec 13* lays down the penalty for unauthorised use.

Allied emblems include 'Red Crescent', 'Red Lion and Sun', and the emblem of the 'Swiss Confederation.'

As per law, the use of any design or wording resembling or likely to be mistaken for any of the above emblems, is subject to penalty. The punishment may take the form of a fine (upto Rs.500/-), as well as forfeiture of the goods upon or in connection with which the emblem, designation, or wording was used by that person.

Despite the unambiguous status of the law in regard to the Red Cross emblem, violations are extremely common in India. Private doctors use it at their place of residence, clinic, or on their vehicle, while chemists use it on their business premises, hospitals use it on their ambulances, and public transport buses use it on their first aid boxes. Such misuse may not be unethical, but it is certainly illegal. However, it is impossible to take cognizance of such widespread abuse, and therefore many cases go unpunished. Moreover, the abuse of the Red Cross emblem may not be deliberately done, but may arise out of the basic misconception that the emblem can be legitimately used by all medical professionals. Even the lay public invariably associates the emblem of the Red Cross with doctors, chemists, pharmacists, ambulances, first aid boxes, and nursing homes, and in fact anything that is connected with health services. There is therefore a dire need to educate the lay public as well as medical professionals, before punitive action is contemplated. [2]

MEDICAL ETHICS

Medical ethics deals with moral principles which should guide the members of the medical profession in their dealings with each other, with their patients, and with the state. A survey of the actual texts of the various 'Oaths' as applicable to medical practitioners, can serve as an introduction to medical ethics.

The general principles mentioned in the **Hippocratic Oath** were brought up-to-date by the World Medical Association several years ago. The modernised versions of the Hippocratic Oath comprise the **Declaration of Geneva** (as adopted by the third General Assembly of the World Medical Association at Geneva, Switzerland, in **September 1948**), and the **International Code of Medical Ethics** (as adopted by the fourth General Assembly of the World Medical Association held in London, in **October 1949**)[3].

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Each country has its own code of ethics, usually modelled on the lines of the International code. In fact, India can boast of an oath proposed by **Charaka**, roughly 4700 years ago ![4]

The reader is advised to consult the *Annexures* at the end for a detailed elaboration of the important 'Oaths' and 'Codes' in relation to the medical profession.

MEDICAL ETIQUETTE

Medical etiquette refers to the conventional laws of courtesy observed between members of the profession.[3] Every medical practitioner should treat his colleagues in the same way as he would have them treat him i.e., with courtesy and grace.

PROFESSIONAL MISCONDUCT (INFAMOUS CONDUCT)

Any act or behaviour of a doctor which is considered disgraceful or dishonourable by his professional colleagues of good repute, amounts to **professional misconduct** or **infamous conduct**. Such cases, when brought to the notice of a State Medical Council, will be examined to determine if the doctor is guilty, and if so, to decide on the punishment. The procedure of enquiry is mentioned on page 12.

It is not possible to enumerate all the situations in which a doctor may be charged with professional misconduct, but the general categories of serious professional misconduct include the following:

I. **Abortion :** Illegal termination of pregnancy is considered as an act of professional misconduct. Even after the reforms in law brought about by the *Medical Termination of Pregnancy Act 1971*, criminal abortions are still performed. It is imperative that termination of pregnancies must be carried out in a proper institution which is officially recognised, and it should be conducted by competent, qualified doctors in the prescribed manner whenever the need arises.

II. **Adultery:** A medical practitioner must maintain the highest standards of moral integrity. He should not misuse his position to commit adultery with his patients or their relatives or attendants.

III. **Alcohol:** Attending to patients under the influence of alcohol is strictly prohibited.

IV. **Addiction:** By virtue of his profession, a medical practitioner has easy access to various kinds of drugs. He can be punished for professional misconduct if he resorts to drug abuse.

V. **Advertising:** The rigid attitude of the past towards advertisement of one's professional skills and ability is gradually being replaced by more liberal views. This change in attitude has become inevitable because of the increasing impact of the media on the community, increasing level of education, and heightened awareness of the lay public about medical matters.

Doctors are fully entitled to participate in this information transfer, as long as it is not intended to confer personal professional advantages. Compared to the past when anonymity was absolute, the participation of medically qualified people on radio and television programmes, and in newspaper articles has become an everyday occurrence.

However, canvassing for patients is wrong and unacceptable, whether it is done by word of mouth, or in writing. It is also not correct on the part of a doctor to advertise a change of consulting hours through a public notice or in a newspaper. This should only be done by an individual notice to the existing patients, or by displaying a note at the doctor's office or con-

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sultation room. The scale of professional fees must never be disclosed in public. Name boards must be modest in size, colour, and lettering. Personal achievements in surgery or medical treatment must never be advertised.

VI. **Association:** Association with unqualified persons to promote one's practice is perhaps still in vogue, but it indubitably amounts to professional misconduct. Similarly, engaging unqualified persons in technical positions is undesirable and unethical. *Dichotomy* or *fee splitting* is also considered highly unethical. Dichotomy refers to receiving or giving commission to colleagues, pharmacists, manufacturers of drugs, etc., for professional favours.

A doctor is expected to provide the best medical treatment available for his patients. If he is not competent or qualified to handle a particular case, then he should refer it to the specialist concerned. It is objectionable to ask for a share in fees from the doctor to whom the case was referred. By fee-splitting, the doctor is receiving fees for work that he has not done, and hence it is unethical[5].

Some other practices bearing the risk of disciplinary proceedings include:

- Ownership of a shop selling pharmaceutical drugs, or medical or surgical appliances.
- Assisting unqualified persons (i.e., without recognised qualifications) in the treatment of patients. This is referred to as *Covering.*
- Associating with bodies or societies comprising unqualified persons who are engaged in "training" health professionals.
- Violation of the provisions of the Drugs Acts in vogue.
- Performing surgical operations when they are not required or indicated.
- Issuing false, misleading, or fraudulent medical certificates.
- Withholding information about notifiable diseases from public health authorities.
- Disclosure of professional secrets (*vide infra*).
- Refusal to render professional service on grounds of religion, nationality, language, or community.

Some of the important examples of acts amounting to professional misconduct can be remembered as the 6 A's : Advertising, Association, Adultery, Abortion, Alcohol, and Addiction.

CONSENT

Consent has been defined to mean "voluntary agreement, compliance, or permission for a specified act or purpose". **Indian Contracts Act,** *Section 13*, states "two or more persons are said to consent when they agree upon the same thing in the same sense."[3] To be legally valid, the consent that is given must be intelligent and informed, i.e., the consent must be given after understanding what it is given for, and the knowledge of risks involved. Medical practitioners must always remember to obtain explicit consent from a patient before examining or treating him. If they treat or operate on a patient without express authority to do so, such treatment or operation will be deemed to be intentional interference with the patient's body without legal sanction. This in turn amounts to assault and battery for which the patient will be entitled to recover damages.

Consent may be either *expressed* or *implied.* Expressed consent may be *written* or *verbal.* Implied consent is indicated by the demeanour and behaviour of the patient and is adequate for routine general examinations, but when special examinations such as vaginal examination, rectal examination, etc., are required, expressed consent must be obtained. For more complicated diagnostic procedures like endoscopy, or for surgical procedures, written

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consent is essential. In hospitals, expressed consent (verbal or written) should preferably be obtained in the presence of a disinterested third party such as a nurse or a receptionist. As far as possible, consent must be obtained after explaining the nature and consequences of the treatment procedure being contemplated. This is termed *informed consent*.

The consent given by any person must necessarily be voluntary and free. Consent obtained by force, fear, or fraud is invalid. Though for routine examinations and treatment a broad based consent (*blank consent*) may suffice, for special operative procedures, specific informed consent in written form is necessary. Consent may not be deemed necessary in the following situations:

i) the patient is in coma and needs emergency treatment.
ii) the patient is a child admitted as an emergency case, and the parents are not immediately accessible.
iii) when a medico-legal case is referred by a court of law for examination.

Consent is to be obtained from the following persons for examination and treatment:

a) Conscious, mentally sound adults.
b) Children above 12 years of age.
c) The spouse of a person who is to undergo sterilization, termination of pregnancy, etc., (not always essential legally).
d) The parent or guardian of a child who is less than 12 years of age.
e) Permission of *loco-parentis* (e.g., headmaster of a residential school) may be obtained when it is an emergency situation, and the consent of the parents concerned is not easily obtainable.

Mentally unsound persons are not capable of giving valid consent. The legal guardian must provide the same. Consent obtained for an illegal procedure is necessarily invalid.

PROFESSIONAL NEGLIGENCE (MEDICAL MALPRACTICE)

Professional negligence refers to a breach of duty leading to liability for damages. This breach of duty may be the result of failure to do something (omission) which a reasonable man under the circumstances would do, or doing something (commission) which a reasonable man would not do. An action for medical malpractice is one for negligence, i.e., the failure of a doctor to perform a known duty owed to his patient. It does not differ from any other action for negligence, except that it requires evidence of a stronger nature than is required for most negligence actions.[6] Malpractice may result either from the doctor's lack of knowledge and skill, or from negligent failure by the doctor in exercising due care in the performance of a procedure.

Every medical practitioner is required to possess reasonable medical knowledge, and to exercise reasonable skill and care in the treatment of his patients. This does not however imply that he should possess the highest degree of skill or knowledge. He is only expected to possess such skill and knowledge as possessed by ordinary competent men practicing medicine at that time under similar circumstances and conditions. He must therefore merely follow the prevalent standard procedures, and methods of diagnosis and treatment. No doctor is expected to possess all currently available medical knowledge, nor is he expected to be able to apply all known diagnostic and therapeutic techniques. But a doctor of a particular standing as regards grade and experience, is expected to have a standard knowledge and capability commensurate with his professional status. Thus a house surgeon is not expected to possess the same skills as a consultant surgeon, but at the same time he is expected to provide a level of medical care which is within his competence.

The elements of a malpractice action generally comprise the following:

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1. Existence of a *duty* of care.
2. Failure to exercise such duty of care (*dereliction*).
3. Causation of *damage* to the patient on account of dereliction. The damage which results must have been reasonably foreseeable.

Professional negligence of doctors is punishable under *Sec 304-A* of the **Indian Penal Code**. According to this section. "Whoever causes death of any person by any rash and negligent act not amounting to culpable homicide shall be punished with imprisonment for a term which may extend to 2 years, with or without fine".

1. **Duty of care:**
   A doctor charged with negligence must be shown to have been under a duty of care to the person complaining of negligence. If the doctor was not under a duty of care to the complainant, he cannot be charged for the negligence even though his conduct has caused damage to the complaining party.

   This relationship may be formed easily, and by no means depends on any formal acceptance of a patient by a doctor. Even in an acute emergency, once a doctor approaches an ill or injured person with the object of assisting him, a completely valid relationship is built, not withstanding the fact that the patient may be unconscious and quite unaware of the doctor's presence. The fact that no payment is offered or expected makes no difference to the duty of care. However, when a doctor acts as a "Good Samaritan" and helps an injured person in a roadside accident, he will not be bound by duty of care. When a doctor examines a patient for some purpose other than providing advice and treatment, no contractual obligation is established and therefore no duty of care exists, for e.g., medico-legal examination for disability, drunkenness, etc.

2. **Damage:**
   'Damage' is the injury or disability suffered by the patient. The failure to exercise a duty of care must lead to actual damage in order to be actionable. However negligent the doctor might be, a patient cannot sue him for negligence if no damage has occurred. It should be proved that the breach of duty was the real cause of damage complained of and such a damage was reasonably foreseeable.[7]

Case Histories

A patient was operated on for the delivery of a child. After being discharged, she complained of pain and distress. She was admitted to another clinic and an x-ray was taken which revealed a pair of forceps in the abdomen. Subsequent surgical intervention revealed a devitalised gut along with the pair of forceps. About three feet of the gut had to be resected. The intestine had entwined itself around the forceps and its blood supply was affected. The National Consumer Disputes Redressal Commission awarded a compensation of Rs.20,000/- in addition to the expenditure incurred for the second operation.*

An employee of Philips India fell ill suddenly and the company Medical Officer treated him though he was not able to make a correct diagnosis. The patient later died of small pox. The Bombay High Court did not consider the doctor negligent as the duty cast on the company doctor was not higher or lower than the duty of an average doctor towards his patient. The doctor in this case had exercised the ordinary degree of professional skill.**

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* ***Mrs. Madhuri** Vs Dr. Rajendra and others. National Consumer Disputes Redressal Commission, New Delhi. Appeal 600 (1993). Consumer Protection Reporter 1996; 10 (3):174.*

** ***Philips India Ltd.** Vs Kanju Punnu. Accident Claims Journal 311, 1975. All India Reporter 1975; 306:77. Bombay.*

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Errors of judgement either in diagnosis or treatment, will not constitute negligence as long as it is proved that the doctor had exercised minimum reasonable standards of skill and care. A doctor cannot guarantee to provide the best possible care, but only care that is consistent with his professional status.

The term '*damages*' refers to monetary compensation that is intended to restore the injured patient as far as possible, to the state he was in before he suffered from a negligent act. Though money cannot be expected to restore a wrongly amputated limb, it could help to compensate for the loss of wages in the case of a patient who can no longer pursue his trade. Damages are categorised into 'general' and 'special' varieties. General damages are awarded for more subjective effects of injury, such as pain and suffering, loss of enjoyment of life, loss of function, etc. Special damages are those which can be more exactly computed, for e.g., cost of treatment, loss of future income, expense of care for a badly disabled person, etc.

3. **Reasonable foreseeability of damage**

It is not every failure to exercise a duty of care that renders the doctor liable. The damage has to be foreseeable. One would have to take into consideration whether a reasonably competent doctor under the same circumstances would have foreseen the injury. If not, the doctor would not be liable for negligence.

For example, dyspepsia (impaired digestion) may be an early symptom of serious illness like gall bladder disease, peptic ulcer, or stomach carcinoma, but often no organic lesion is found in the early stages. Before the doctor orders special (and expensive) investigations like sonography, barium studies, endoscopy, mucosal biopsy etc., he may advise a detailed study of stool to exclude parasitic infestation. It is possible that stomach cancer may be missed or diagnosed late, but is certainly not negligence in this case, as the doctor's approach was based on sound judgement.[7]

A doctor will be considered negligent in the following circumstances:-

i. If the doctor's professional performance falls below the standard of a reasonably competent medical practitioner.
ii. If there is overt evidence of negligence in diagnosis, treatment procedure (including administration of medicines), surgical operation, etc.
iii. Evidence of failure in undertaking all reasonable precautions.
iv. Evidence of any other form of negligence in providing care and treatment.

Calculated Risk Doctrine

In certain management manoeuvres, there may be a certain amount of inevitable risk. For example, amniocentesis is associated with a 1 per cent risk of abortion. In a cesarean section, there is 0.1 percent chance of maternal mortality. In such cases, a charge of negligence will not stand against a doctor if proper care has been taken to avoid the risk. Justification for the chance taken for the risk, and proof of preventive measures taken can constitute adequate defense on the part of the doctor.[8]

Novus Actus Interveniens (An Unrelated Action Intervening)

This usually applies to cases of assault and accidental injury. It is applicable in a situation where the cause of disability or death passes from the original incident for which the doctor was responsible, to the later negligent action of some other person. However, it would be difficult to prove in cases of disability or death, whether it was the result of the original negligent act, or due to the subsequent incident.

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Res Ipsa Loquitur (The Thing Speaks For Itself)

It is a situation where the facts of the case itself are sufficient to justify the conclusion that most probably the defendant (doctor) was negligent, and the negligence caused the injury. It is a reasonable conclusion from the circumstances of an unusual accident, that it was probably the doctor's fault. The plaintiff (patient) does not have to prove that the act was negligent as it could be nothing else. It is for the doctor to prove if he can, that the negligence was not his doing. Examples : amputation of wrong limb or digit, leaving a swab or instrument in the patient after operation, operating on the wrong part, etc.

Contributory Negligence

Contributory negligence refers to any unreasonable conduct or negligence on the part of the patient which has contributed to the causation of damage alleged, although the doctor has also been negligent. This may occur when, for example, the patient interferes with his dressings and induces infection, or removes a plaster cast or bandage, or more commonly, fails to attend the follow-up clinic. In claiming that there was contributory negligence, the burden of proof will be on the doctor to show that it has happened. If it is proved, the compensation payable will be reduced.

Corporate Negligence

Sometimes a patient's care is affected by malfunctioning equipment, inadequate accommodation, incompetent staff, etc. In these cases, if harm results to the patient, the hospital becomes liable for negligence. This is referred to as corporate negligence.

Products Liability

Sometimes, injury or death of a patient may result from the use of faulty, defective, or negligently designed medical or surgical equipment, or due to the use of a drug that is adulterated, contaminated, or of inferior quality, or due to the lack of accurate guidelines in the use of any equipment or drug. In such cases, the manufacturer concerned becomes liable for harm resulting to the patient. This is referred to as products liability.

However, the manufacturer will not be liable in the following situations -

- if the doctor or hospital misused an equipment or medical product.
- If the instrument was functioning well at the time of supply, and was not defective.
- If the instrument malfunctioned due to improper use, or due to wear and tear, and was not serviced regularly, or replaced in time.

Therapeutic Misadventure

This is applicable in those situations where a diagnostic procedure or treatment carries inherent risks of complications. The employment of some investigative procedures or therapeutic measures by a doctor involving considerable risk and compulsion, in the absence of an alternative option (e.g., use of anticancer drugs), will not provide grounds for medical negligence, unless the doctor failed to draw the attention of the patient to the risk at the time of obtaining consent.

Differences between Civil Negligence and Criminal Negligence (Table 2.1)

Vicarious Liability

As per law, a man may become liable to pay damages for an act of negligence committed by his servants or agents in the course of their employment or agency. This is called vicarious responsibility or vicarious liability, or as the Latin phrase goes "respondeat superior", meaning *let the superior be responsible*, or *let the master answer.* For a person

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Table 2.1 Differences between Civil Negligence and Criminal Negligence.

Civil Negligence	Criminal Negligence
1. Simple absence of care and skill	Gross carelessness and scant regard for patient's welfare.
2. No special violation of law	Willful, wanton, or even culpable negligence.
3. Consent of patient amounts to a good defence	Consent of patient not adequate defence.
4. Tried by civil court	Tried by criminal court.
5. Contributory negligence can be cited as a defence in court	Contributory negligence does not constitute defence in court.
5. Liable to pay damages	In the event of death, punishable under Sec. 304 (A) of the IPC.

to become vicariously liable for the act of another, it must be proved that

i. the person was his servant or agent, and
ii. the negligent act was done in the course of carrying out his duties.

A surgeon may be vicariously liable for the negligent act of his assistant when he is in charge and directing the operation. However, the action of an anaesthetist is not imputed to the surgeon, unless the surgeon issues specific orders to the anaesthetist.

PROFESSIONAL SECRECY

During the course of treatment, a patient may reveal matters of a personal nature to his doctor. The doctor is obliged to maintain the secrecy of all such information, except when he is required by law to divulge it, or when the patient has consented for the disclosure. The doctor is liable for damages if he discloses without valid grounds, any such information which the patient may reasonably claim as secret or confidential. The following examples can serve as guidelines:

1. A doctor should not disclose any secret information about his patient or his private life which he learns in course of his examination, to a third party.

2. Without the consent of the patient, no information about his illness, or any matter about his family can be disclosed, even to the patient's spouse or employer.

3. In the case of domestic servants, even if the master pays the fees of the doctor towards the treatment of the servant, the doctor should not disclose any information to the master about the nature of the illness, without the patient's consent.

4. Information about the nature of illness of a minor child, insane person, or intoxicated patient, may be disclosed to the guardian of the patient. But in the case of a mentally sound major patient, nothing should be disclosed to his parents or any other relative without his consent.

5. While treating a minor inmate of a hostel or a boarding house, the doctor may disclose relevant

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information about the patient to the warden or superintendent. But if the patient is a major, he cannot do so without the consent of the patient, unless the matter poses risk to the other inmates of the hostel.

6. In the course of treatment of a convicted prisoner in a jail, the matter relating to his illness may be conveyed to the jailor or the superintendent of the jail. But in the case of a prisoner under trial, or a person in police custody, this cannot be done without his consent.

7. When a person approaches a medical practitioner for a medical certificate or a report, it may be taken as implied that he has given consent for the disclosure of all information about his illness. Hence, in such a situation, disclosure of relevant information about the person, will not amount to breach of trust.

8. While reporting a case in any scientific journal, the doctor should take care that the identity of the patient is not disclosed.

Every doctor must respect the information he obtains from his patients. He is not expected to disclose these secrets under ordinary circumstances. However, there are certain circumstances in which a doctor is bound by law to divulge information, even if it is of a confidential nature. This is referred to as *privileged communication.*

Privileged Communication

A medical practitioner cannot withhold professional secrets in those cases where he has a statutory duty to notify births, deaths, infectious diseases, etc., to the public health authorities. On certain occasions, he has a moral duty to protect the interests of the community or the public, and in doing so, if he divulges the secrets of his patient obtained in the course of his professional examination and treatment, he will be absolved from legal liabilities. The following examples are self explanatory :

1. A medical practitioner has a duty to inform the warden of a hostel, if any boarder is suffering from venereal or other communicable or infectious disease.

2. He must inform the railway authority if he finds that a particular engine driver is colour blind, and he does not wish to change his employment, although he is persuaded to do so.

In such cases, the communication (if made bonafide and without malice), will be deemed to be privileged by the circumstances. However, it should be made only to persons directly concerned, otherwise he may be penalised.

A privileged communication is therefore defined as *a statement made by a person who has an interest to protect, or a legal, social, or moral duty to perform to another person having a corresponding interest, even though such communication may under normal conditions, amount to defamation or slander.*

ORGAN TRANSPLANTATION

With the introduction of the **Transplantation of Human Organs Act 1994**, an individual above the age of sixteen can volunteer while he is alive, to donate tissues from his body to any needy patient, provided he is of sound mind. His consent should be obtained in writing, after explaining to him the details of the procedure to be followed, including the risks involved if any. If the risk involved is of a serious nature, organ donation should not be accepted even if the donor is willing.

Cadavers constitute a good source of tissue for transplantation. During the course of his lifetime, a donor can give his consent for the removal of tissues from his body after his death. Such a consent must be given in writing and two disinterested

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persons must bear witness. Even when the consent has been given by the deceased during his life, permission must also be obtained subsequently from the person who is in possession of the dead body. It is possible that the person in possession of the body may overlook the wishes of the deceased, and not agree to donate the tissues. Occasionally, the person in possession of a dead body may give permission for removal of tissues from the body, in the absence of explicit consent of the deceased while he had been alive. This can be legally valid, provided there is no reason to believe that the deceased had expressed a desire to the contrary while he was alive. There should be no objection from the surviving spouse or near relatives.

Normally, the right of possession of a dead body lies with the surviving spouse or the next of kin. In case of death in a hospital, the hospital is the legal possessor until someone with a better claim to possess it turns up. If nobody turns up to claim the body for 72 hours after death, the body may be disposed of by the hospital authorities. If the deceased had left any directions about the manner in which the body is to be disposed of, or the manner in which the tissues are to be used, such directions must be respected.

Any transplant must be effective in its own right to be useful. A donation from a living person is more likely to succeed than that from a dead donor, and in the latter case, success will be inversely proportional to the length of time between death and donation. The propriety of live donation of organs for transplantation depends on the following factors:

a) there should be a reasonable prospect of the transplant being successful

b) the benefit to the recipient must far outweigh the likely detriment to the donor.

c) it should fall under the concept of *potential donations*.

Potential donations are of three types:

i. Donations of tissues that are readily replaceable (e.g. blood). These do not cause any serious problems.
ii. Donations of organs that are not essential to the life of the donor.
iii. Donations of one of the paired organs, the other one being capable of maintaining life, as long as it is healthy (e.g., kidney).

Consent to such donations should be properly obtained, after giving a complete explanation.

The ultimate success of a transplantation rests on the quality of the donated organ, and this depends on the *warm anoxic time* to which it is subjected. This is the time between the cessation of arterial oxygen supply and the refrigeration of the isolated organ. Living donations offer two major advantages. Firstly, sufficient time is available for a full appraisal of the case, and secondly, the warm anoxic time can be reduced almost to zero. However, in the case of cadaver donation, the kidney must be removed from the dead body within 30 to 60 minutes of circulatory cessation, and in general must not be stored for more than 12 hours. **Simpson** has described the moment of death as "the starting pistol for a race to save life".

The dramatic improvement in the success of transplant surgery that has taken place in recent years has been largely due to the approximation of the cadaver to the living donor through the medium of *beating heart donor*. This concept has become possible with the use of sophisticated machines for keeping the respiration and circulation going on in a person after he is declared dead. There are functional regions in the brain that vary in their resistance to lack of oxygen, the most sensitive being the cortex, followed by the thalamus, and finally the brain stem which regulates the basic functions of the body and in particular, the respiratory and

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vasomotor centres. There may be varying degrees of 'brain death' ranging from intellectual deterioration to suppression of all functions, (except the capacity to remain alive.) This is known as *persistent vegetative state (PVS).*

In any discussion of death, it is essential to appreciate that death is an absolute concept ; the body is not dead unless the whole brain is dead. The brain stem is not only the controller of life, but also the most important part of the brain which is relatively resistant to hypoxia. For this reason, the common expression '*brain death*' has now been replaced by the unambiguous term '*brain stem death*'. It is now generally accepted that brain stem death can be diagnosed while the heart is still beating. However, some stringent criteria are laid down. They are called the *Harvard Criteria.* These include the exclusion of drug overdose, hypothermia, or metabolic disorder as a cause of deep coma, and establishing that the cause for the coma is irremediable structural brain damage. This is followed by tests to confirm that all brain stem reflexes are absent, and in particular to establish under very standardised conditions that no respiratory movements occur, when the patient is disconnected from the mechanical ventilator. It is recommended that the tests be repeated after a reasonable interval (usually 6 hours). The standard practice is that the diagnosis should be made by a minimum of two doctors, and it is mandatory that they do not form part of the team concerned with transplant operation. If brain stem death is declared after all the conditions have been met, the patient is considered unequivocally dead, and the decision as to stop or continue ventilation rests upon whether the body is to be used as an organ donor. (**"Organ Transplantation" & "Brainstem Death"** have also been discussed in **Chapter no. 6**).

CONSUMER PROTECTION ACT, 1986

The Consumer Protection Act received the consent of the President of India on **24th December 1986.** It has undergone two amendments, one on **18th June 1993** and another on **27th August 1993.**

The purpose of the Act is to make sure that business and industrial organisations are obliged to satisfy the needs of consumers by producing quality goods and services at reasonable prices. In the corporate sector, which is a form of organisation producing goods and services, the emphasis is also to give proper after-sales service, and provide a mechanism for satisfactory response to grievances and problems of the consumer. The Act seeks to provide a cheap and speedy remedy to the ordinary consumer who is unable to get involved in a costly and time consuming litigation in a court of law in search of redressal for his problems. The Act covers all private sector, corporate sector and public sector enterprises. The service rendered by doctors has been explicitly brought under the purview of the Consumer Protection Act in 1995. However, services offered by Government hospitals which provide absolutely free treatment are not covered by the Act.

The District Consumer Forum, the State Consumer Redressal Commission, and the National Consumer Redressal Commission (all courts of law, with certain modifications established for the purposes of the Act), are agencies for redressal of disputes in consumer related cases and matters connected therewith.

The **consumer court** is a *three tier* structure. The *lowest tier* is the District Forum. It is established by the State Government with prior approval of the Central Government in each district of a state. The district forum has three members. One among them is the President, who must be a district judge or a retired district judge,while the other two are eminent citizens, one of whom should be a lady. These fora have a jurisdiction to entertain claims for compensation upto Rs.5 lakhs. The *second tier* is the State Consumer Redressal Commission. It

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comprises of three members. The President has to be a High Court Judge or a retired High Court Judge. Two other members are eminent citizens, one of whom has to be a lady. The State Commission hears appeals from district forum and has jurisdiction for consumer compensation claims between Rs.5 lakhs and Rs.20 lakhs. The *topmost tier* comprises the National Consumer Redressal Commission presided over by a retired judge of the Supreme Court with four other members, one of whom has to be a lady member. The National Commission hears appeals from State Commissions, as well as consumer complaints seeking compensation of over Rs.20 lakhs.

Certain situations such as services rendered free of cost, or purchase of goods or services which are not meant for personal consumption, but for business purposes, will preclude a claim from the purview of the Act.

Procedure

The procedure under the Consumer Protection Act is very simple. There is no court fee. The complainant (consumer) has to submit his complaint to the members of the concerned consumer court. The consumer court issues notice to the opponent by sending copies of the complaint, and asks for a reply within 30 days. If the opposite party disputes the complaint or fails to represent his case, a hearing is held. It is not necessary for the parties to be represented by lawyers. The Act allows only one adjournment. Within 90 days an order is to be given, unless the goods in question are to be sent to an approved testing laboratory, in which case the period is extended to 150 days.

The complaint has to be filed within two years of the cause of action, unless the court condones the delay for special reasons.

The Act also allows provision for establishing consumer protection councils at the central and state government levels. These councils will have members selected by the government, from the concerned departments of the government, and also non-official members representing such interests as may be prescribed. The objectives of these councils are to promote and protect the right of the consumer.

The Supreme Court verdict of **Nov.1995** defined the treating doctor as the provider of medical service, and the patient as a consumer of such service.

Criticism

The Consumer Protection Act has shattered the age old relationship between doctor and patient, which is based on mutual trust and faith. In order to protect his own interests, the doctor may prescribe expensive tests and investigations which in the ordinary course of treatment may not be required. Though aimed at penalising erring and negligent doctors, the Act has pressurised the medical profession to resort to defensive attitudes while dealing with patients. The Consumer Fora which try such cases, do not have a medical practitioner on their panel who can screen medical complaints. Without expert guidance, these consumer courts raise the possibility of jeopardising the principles of justice.[9]

PREVENTION AGAINST NEGLIGENCE

The vast majority of medical negligence cases fall under a few common causes, and therefore it would be advisable to take precautions against common pitfalls. *Prevention is better than cure.* This dictum perhaps was never as valid elsewhere as it is in a case of negligence.

1) **Do not criticise another doctor** : A physician must remember that one of the most common causes of negligence arises out of criticising adversely the professional conduct of another physician, without knowing the full facts[10]. It has

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been observed that quite often patients go from doctor to doctor asking for an opinion regarding investigations or treatment adopted by another who is either a family physician or the medical officer at the place of work. The patient is perhaps just seeking psychological reassurance, but a casual remark by any doctor can create complications by shaking the patient's confidence in the doctor whom he has originally consulted.

2) **Employ qualified staff and associate with good partners**: The doctor must always remember that he is responsible for the acts of omission/commission of his assistants, non-technical staff, and partners in profession during the course of treating the patient. Therefore selection must be done very carefully, and only those who are properly qualified should be employed.

3) **Update your knowledge** : A doctor must keep abreast with the latest developments with reference to his profession and its practice.

4) **Keep accurate and complete records** : Human memory can fail, but accurate and detailed medical records will never fail a physician in his hour of need, even after a considerable lapse of time. The doctor must keep up-to-date records, complete with respect to:-
   a) History - including negative and positive in formation.
   b) Present illness - in the patient's own words as far as possible.
   c) Physical examination - both subjective complaints and objective findings.
   d) Course of investigations - advised, executed, and their results.
   e) Impressions about the case-before and after diagnostic procedures, and the diagnosis.
   f) Treatment adopted - a complete day-to-day record.
   g) Any consultation advised or references made to specialists.

   Any refusal on the part of the patient regarding treatment, or diagnostic investigations, etc., or discontinuing treatment against the physician's advice, should be recorded and the signature of the patient obtained against the statements.

5. **Take valid consent**: Consent of the patient must be obtained before starting an examination, diagnostic or investigative procedure, or the treatment. Failure to obtain valid consent before commencing such measures may make a doctor liable for assault and battery. For the consent to be valid, it must be *informed*, with the patient being informed of the relevant facts regarding the procedures being adopted or to be adopted, including the risks involved. Whether the consent is implied or expressed, verbal or written, is immaterial. However, it is better to obtain written consent for certain special procedures of diagnostic and therapeutic value.

6. **Reasonable skill and care** : A doctor should take all due care and use reasonable skill within his knowledge. When in doubt it is good to take a second opinion or consult a specialist, and adopt the necessary diagnostic procedures available. While ensuring reasonable skill and care, do not guarantee results even in an implied manner.

   A doctor must ensure that all the instruments used by him are maintained well and sterilized whenever necessary. He should treat his patient as long as it is necessary, and not withdraw from the case without prior notice to the patient.

   If more than one doctor is involved in treating a case, good communication is essential and of benefit, both to the patient and to the doctor.

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7. **Guard against therapeutic hazards**: Before commencement of treatment, the doctor should enquire about the past history of the patient for any adverse drug reactions. Though he may have taken enough precautions to inject a test dose, he should be prepared in advance to handle a case of adverse drug reaction.

CONCLUSION

Medical ethics is an integral part of medical practice. Medical practitioners are constantly called upon to make ethical decisions when dealing with patients. Unfortunately very little learning in ethics takes place in the teaching institutions. Not even 10% of the medical colleges have medical ethics as a separate subject of learning.

It is often thought that ethics is assimilated by a kind of 'diffusion' or 'osmosis' from the teachers[11]. A great deal can be 'caught' by observing the teachers. Teachers have a great responsibility to be role models. It is also true that, a lot can be learnt from systemic teachings of the principles of medical ethics and their applications. Ethics is such an integral part of medicine that we can never have too much of it.

Box 2.1 Case Studies in Medical Negligence.

Case No. 1
AKH vs Bombay Hospital Trust, 1992

AKH was admitted to Bombay Hospital on 19/08/91 for an operation on his left hip. Dr.KTD operated on him the same day, but AKH died the next day at 5.30 a.m. A law suit was filed by his wife and children. The complainants alleged carelessness and negligence while treating the patient, as a result of which the patient died. The surgery had been scheduled for 10 a.m., but due to the late arrival of the doctor, AKH was operated only at 1 p.m. It was further alleged that after the operation, AKH did not regain his consciousness till late in the evening. He was restless and complained of pain and continuous bleeding from the site of operation. The wound bled continuously till the death of the patient, the next morning. Further allegations of the complainants were that no senior doctor attended upon AKH, and no treatment was given for the continuous bleeding except for one bottle of blood, which was not sufficient.

The case was not contested by the opposite party. The verdict was in favour of the complainants, and a compensation to the tune of Rs.7,00,000/- was ordered to be paid.

Case No.2
SGC vs CW Hospital, 1994

SGC filed a complaint alleging negligence in a case of chronic renal failure requiring renal transplantation. The complainant had brought his son R to CW Hospital, Miraj, for treatment. The complainant was informed that R needed a kidney transplantation, for which he was to be put on dialysis. In order to perform dialysis, a needle was inserted in the groin and this needle was left there with the application of adhesive tape. He was told that the needle was to be kept there, as he needed frequent dialysis. The complainant alleged that the same needle was kept in the groin and dialysis was performed three to four times. Blood and pus were exuding from the injury where the needle was inserted. When the adhesive tape was removed subsequently, it was found that the injury caused due to insertion of the needle was infected. Ultimately, the right leg had to be amputated. Even after this, the infection could not be controlled and the patient died about a month after the amputation.

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The opposite party did not appear in court, and after hearing the arguments of the complainants, and the witnesses, the court ordered a compensation of Rs.2 lakhs to be paid.

Case No.3
VA vs L Hospital, 1991

Mrs.VA aged about 26 years, filed a complaint under sec 21 of the Consumer Protection Act, 1986. The complainant alleged negligence on the part of doctors who conducted MTP without proper examination, and diagnosing a case of cervical pregnancy, conducted a hysterectomy. This , she complained, led to mental disturbance, psychological depression with impairment of physical health, and robbed her of her ability to bear children. But the complaint was dismissed, as the Consumer Court held that the doctors concerned had acted with due care, circumspection, professional skill, and competence. During the operation, the lower uterine segment was opened and all the products of pregnancy were removed. It was seen that the products of pregnancy had attached to the wall of the upper part of the cervical canal and that it had grown into the lower uterine segment and also got adherent to the uterine scar left by the previous caesarean section. It was also seen that her urinary bladder had become adherent to the uterine scar. The bleeding was not controlled even after evacuation and suturing of the lower uterine segment. Every attempt was made to preserve the uterus. However, the complainant's condition deteriorated very fast and she was bleeding profusely. It was under these circumstances that an emergency hysterectomy was performed to control the bleeding and to save the complainant's life.

The court was convinced that there was no negligence of any kind on the part of any of the concerned doctors. In fact it was only due to the prompt and efficient surgical treatment administered to the patient in the emergency situation, on the discovery of the cervical pregnancy, that her life had been saved.

Case No.4
M vs C Laboratory, 1995

The complainant M had got his blood tested at C Laboratory on 21-07-89, and was told that his blood group was 'O' positive. An entry was made to this effect in his driving licence. Later, when his ailing sister-in-law required transfusion of blood of the same group, the complainant who was then at Madurai rushed to Calicut to donate his blood at N Hospital where his sister-in-law was admitted as an inpatient. When his blood was tested at the hospital it was found to be 'A' positive. This was confirmed by another laboratory subsequently.

The District Forum held that the opposite party had committed deficiency in service, and ordered a compensation of Rs.1000/-, plus Rs.200/- towards expenses.

Adapted from **Nagpal N.** *Compendium of CPA Medical Judgements.* **1996.** Neelam Prakashan, Chandigarh.

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CHAPTER - 3

Euthanasia

"Killing both pain and patient may be good morals, but it is far from certain it is good law".

- Lord Edmund Davies (1977)

"State prohibitions that would force a dreadful, painful death on a rational but incapacitated terminally ill patient are an affront to human dignity".

- Justice J. Cory (1993).

Origin

The word **euthanasia** is derived from the Greek, and means a "good death", though it has undergone considerable distortion over a period of time to assume its present day meaning of "mercy killing". As far back as **300-400 BC**, both **Socrates** and **Plato** accorded moral sanction to euthanasia and suggested that it was permissible in certain circumstances. But today there is a raging controversy all over the world as to its legal standing (evident from the quotations mentioned above), aside from the moral and ethical issues involved.

Synonyms

Mercy killing, Assisted suicide, Physician-assisted suicide, Aid-in-dying, Self deliverance.

Meaning

Euthanasia refers to the infliction of a painless death on an individual suffering from severe, intractable pain or discomfort, arising out of a terminal illness or condition.

Classification

Voluntary - The dying individual consents to the act.

Non-voluntary - The dying individual is incapable of giving or refusing consent, e.g., an irreversibly comatose patient.

Compulsory (Involuntary) - Decision by society to terminate the life of an individual.

Active - Inflicting painless death by an act of commission, e.g., injection of a lethal dose of barbiturates.

Passive - Inflicting painless death by an act of omission, e.g., withdrawal of life support to a comatose patient.

Paediatric - Euthanasia administered to seriously sick or deformed infants.

Geriatric - Euthanasia administered to seriously sick aged individuals.

Battlefield - Euthanasia administered to severely wounded or handicapped individual.

Arguments in favour of euthanasia

The medical ethos of the "sanctity of life" implying the unqualified duty to preserve life at all costs, has come under increasing challenge. There is a

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perceptibly growing movement away from a rigid obligation on the part of a doctor to preserve life "at all costs", toward a more flexible duty to provide good palliative terminal care.[1] Terminal illness is considered best treated by providing optimum conditions for death rather than by the use of increasingly invasive techniques to prolong some sort of existence. A conscious, dying patient can make his own decision, since patient's autonomy is today considered to be the corner-stone of ethical medical practice. To continue to treat in the face of contrary instructions can attract legal liabilities, though the exact position is still not clear-cut in India (*vide infra*). In many Western countries, it is generally accepted that there is no moral obligation on the part of a doctor to preserve life at the expense of suffering, and if in the course of good terminal care, the use of drugs inadvertently hastens death, this is ethically acceptable within the concept of "double effect," i.e., an ill effect is morally acceptable as long as there is a greater, intended good effect from an action. This concept was first given legal sanction in relation to euthanasia by **Justice Devlin** in the *Bodkin Adams case* (1957), when he remarked, "The doctor is entitled to relieve pain and suffering even if the measures he takes may incidentally shorten life."[2]

The possibility of maintaining "physiological life," i.e., the continuation of the body functions by artificial means, while the patient remains unconscious over a period of months or years, has introduced a new dimension into the debate - "quality of life".[3] Clearly, the sustaining of physiological functions with no prospect of recovery of consciousness or contact with the patient's environment has led to considerable debate as to whether or not such support systems should be continued, and who should be responsible for the decision to turn off such systems.

Views vary widely, but there is a substantial proportion of population in countries such as the USA which favours the view that support systems can be switched off by attending doctors with consent from the victim's relatives, in certain cases. The case of *Karen Ann Quinlan* is representative of this. Karen, a 21 year old girl fell into a coma after imbibing an overdose of alcohol and drugs on 15 April, 1975. She passed into "persistent vegetative state" from which recovery was extremely remote. Her parents, both devout Roman Catholics, were initially hopeful of a miracle, but as the months passed by they realised that this was only wishful thinking, and that their daughter was never going to come back to meaningful existence. They requested that life support systems prolonging the futile ordeal be switched off. The doctors attending on the case refused, and the issue was taken to court. In 1976, the New Jersey Supreme Court delivered a verdict in favour of Karen's parents. However, while the support systems were subsequently switched off, artificial feeding was not stopped, and Karen continued to "live" for nearly 10 years before her heart finally stopped in 1985.[4] The case however served to focus medical attention on a topic that had for decades been considered taboo.

Today, not only in the case of terminally unconscious patients, but also in conscious patients (who are suffering from an intractable, painful, terminal illness), euthanasia is being increasingly considered as applicable, provided the patient concerned makes the request, and he is judged to be of sound and rational mind. Withdrawing treatment and allowing a patient to die (passive euthanasia) is therefore not perceived as repugnant anymore in certain situations. In fact in several Western countries, adult individuals over a specified age can execute a *living will* or *advance directive* which entitles him to refuse treatment any time in future in the event of a catastrophic accident or natural disease.[5]

While passive euthanasia has been accorded some semblance of acceptance in certain situations by most people, active euthanasia is in the centre of a

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heated (and often bitter) debate, with vociferous sections of the population in India and elsewhere either strongly advocating or condemning its practice. Despite its unresolved status, active euthanasia is being employed without serious legal difficulties in some countries, e.g., Holland, USA, Sweden, etc. Some countries have virtually legalised active euthanasia if performed by a physician under strict guidelines (physician-assisted suicide), e.g., Columbia. Some parts of the world have enacted legal statutes, e.g., **The Death with Dignity Act (1994)** of the State of Oregon, USA, and The Northern Territory of Australia, which was however overturned by the Australian Senate in 1997. Holland practices physician-assisted suicide openly ever since the Royal Dutch Medical Association officially endorsed it in 1984, and the parliament approved the practice, provided it was done within a framework of specified guidelines.[6]

Arguments against euthanasia

The opponents of euthanasia (particularly *active euthanasia*) state that there are moral, religious, and ethical objections which cannot be ignored. They argue that no one has the right to take away the life of an individual, not even the individual himself. The concept of "sanctity of life" is inviolable and doctors having taken an oath (the Hippocratic Oath) "to preserve life at all costs", cannot justify allowing a patient to die either through active or passive means. It is also true that doctors are not omniscient; there have been several cases where a patient certified as terminally ill, has survived and led a reasonably fulfilling existence for several years with good palliative care. Even if a patient does have only a few months to live, adequate medical care can in most cases serve to make him reasonably comfortable for the remaining period till natural death supervenes. The emphasis therefore should be on improving palliative care, rather than improving methods of terminating life.

A disturbing feature that has come to light in those countries where physician-assisted euthanasia has been accorded legal sanction, is the impatience (and sometimes greed) of the relatives of a dying patient who happens to be affluent, in pressing ahead with "euthanasia" for their own selfish ends.[7] In such cases the patient's "right to die" is subtly converted to a "duty to die", pressure being brought on the hapless victim to accede to the infliction of "painless death".

Evolution of euthanasia

A 20th century chronology of important events relating to voluntary euthanasia and physician-assisted suicide is presented in **Table 3.1**.

Table 3.2 lists some of the important personalities around the world who have been campaigning for the legal acceptance and validation of euthanasia.

Current legal status in India

While euthanasia is not explicitly stated as a criminal offence in the **Indian Penal Code**, it is generally accepted that it is covered either under the offence of attempted suicide (**S.309**), or abetment of suicide (**S.306**). However, judicial opinion acknowledges that euthanasia is a "grey area" in Indian criminal law. This unsettled status has been brought into stark focus in several recent cases, e.g., *P. Rathinam V. Union of India* (**1994**), *Smt. Gian Kaur V. State of Punjab* (**1996**), etc. As per the Supreme Court's view, passive voluntary euthanasia can be visualised as a fundamental right protected by Article 21, which assures the right to privacy, since such right to privacy can be said to encompass the right of a patient to refuse life-saving medical treatment.[8] In other words, the right to "personal liberty" as guaranteed by Art.21 of the constitution includes the freedom to die with dignity. Active euthanasia (voluntary or otherwise) is ofcourse unambiguously viewed as a crime, though convictions have not been brought so far against any individual in India for the commission of such an "offence".

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Table 3.1 A 20th Century Chronology of Euthanasia. (Summary of important events)

Year	Developments
1906	First euthanasia bill drafted in the USA (Ohio), but it does not succeed.
1935	World's first euthanasia society is founded in London.
1938	The Euthanasia Society of America is founded in New York
1967	The first "living will" is written by American attorney Louis Kutner.
1973	The American Hospital Association creates Patient Bill of Rights, which includes the right to refuse treatment.
1974	The Euthanasia Society in New York is renamed the Society for the Right to Die.
1976	The California Natural Death Act is passed which protects physicians from being sued for failing to treat incurable illnesses. Several other states of the USA follow suit with similar legislation.
1976	First international meeting of Right to Die groups.
1978	A Broadway play *Whose Life is it Anyway ?* about a quadriplegic wanting to die is staged for the first time. It goes on to become a massive hit and is followed by its movie version in 1982.
1980	Pope John Paul II issues "Declaration in Euthanasia" opposing mercy killing, but permitting the right to refuse extraordinary means for sustaining life.
1980	Hemlock Society (advocating euthanasia) is founded in California by journalist Derek Humphry.
1980	World Federation of Right to Die Societies is formed in Oxford, England.
1984	The Netherlands Supreme Court approves voluntary euthanasia under certain conditions.
1990	American Medical Association permits physicians to withhold or withdraw treatment from patients who are close to death.
1990	Dr. Jack Kevorkian (Michigan, USA), assists for the first time, in the death of a patient. Over the next 9 years he assists in a total of 130 deaths. He will be tried 4 times before finally being convicted in his 5th trial in April 1999 for 2nd degree murder, and sentenced to 10 to 25 years in prison.
1991	Derek Humphry publishes "Final Exit", a how-to book on self deliverance. It quickly tops the US best-seller list, and is subsequently translated into 12 languages.
1993	A political action committee Oregon Right to Die is founded to write and subsequently pass the Oregon Death with Dignity Act. The Act will take effect only from October 1997 after several unsuccessful attempts to scuttle it.
1996	The Northern Territory of Australia passes voluntary euthanasia law which was drafted the previous year. However, nine months later the Federal Parliament quashes it.
1999	Dr. Kevorkian sentenced to 10-25 years in prison for 2nd degree murder of Thomas Youk suffering from motor neurone disease. The videotape of Youk's death had been telecast on national television, provoking public outrage.

Adapted from **Neera Dahiya.** *Euthanasia and Assisted Suicide.* **1999.** New Delhi.

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Table 3.2 Some prominent *Right to Die* activists.

Name	Country
Pieter Admiraal	*Netherlands*
John Beloff	*Scotland*
Buji Colabawalla	*India*
Lynda Cracknell	*Australia*
Derek Humphry	*U S A*
Ludovic Kennedy	*England*
Jack Kevorkian	*U S A*
Jeanne Marchig	*Switzerland*
Philip Nitschke	*Australia*
Marion Rookhuizen	*Netherlands*
Clemencia Uribe	*Columbia*

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CHAPTER - 4

Identification

"So God created man in his own image. In the image of God created he him".
- Old Testament, ***Genesis 1 : 27***

Identification in the medico-legal sense refers to the determination of individuality of a person. It may be **complete** or **partial**.

In the case of living persons, as well as dead bodies recovered before the process of putrefaction has set in, identity can be fixed with respect to their name, age, sex, occupation, address etc., with the help of their unique features. This is termed **complete identification**. Establishing complete identification is usually the responsibility of the police.

But in cases of mutilated and highly putrefied bodies, as well as in skeletal remains, the police may require the help of a medical officer, or a forensic expert, who with his knowledge of anatomy can help provide certain information such as race, age, sex, stature, etc., by studying the morphology of the body parts. This is termed **partial identification**. Partial identification by a medical officer can provide some clues to the police for further investigation leading to complete identification.

Features like dermatoglyphics (ridge patterns) of the palms and the soles are highly individualistic leading to positive and conclusive identification of mutilated remains. Moreover, with the advent of DNA fingerprinting, complete identification is possible even with a few drops of blood or other body fluids, or a small piece of tissue, hair, bone, etc. This can be accomplished even in the absence of antemortem records of the deceased, since his DNA can be compared with that of his close blood relatives.

The following factors help in establishing the complete identity of a person, either living or dead:

- Race
- Sex
- Age
- Hair
- Anthropometry
- Dactylography
- Poroscopy
- Footprints
- Cheiloscopy
- Rugoscopy
- Tattoo marks
- Scars
- Occupational stigmata
- Complexion & features
- Miscellaneous

RACE

Human beings are usually classified into three major races:

1. Caucasoids
2. Negroids
3. Mongoloids

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Race determination is mainly the domain of physical anthropologists. Due to global shrinkage and increasing incidence of mass disasters such as airplane crashes, etc., their help in identifying victims has become vital in many cases, which has led to the development of a specialised field, **Forensic Anthropology**.

Race can be determined by studying
1. Variations in morphology.
2. **Osteometry**, i.e., measurement of various bones and studying the proportionalities to each other, and
3. Multivariate discrimination function analysis.

Krogman studied the morphological traits of the skull and outlined the essential descriptive morphology of the three main races[1]. He further subdivided the caucasoids into Nordic, Alpine, and Mediterranean groups on the basis of his findings.

Attempts were also made by **Todd and Lindala (1928)**, **Cobb (1934, 1942)**, and **Lewis (1942)**, to differentiate the races on the basis of measurements of different body parts. Due to wide standard deviation, there is considerable overlap in the actual dimensions of various bones among the races, and hence the proportionality of different parameters were studied. This has led to the calculation of various indices such as *cephalic index, brachial index, crural index, inter-membral index, intra-membral index*, etc., (**Table 4.1**). The values of some of these indices for the three races - Asiatic Indians, Europeans, & Negroes are mentioned in **Table 4.2.**

Giles & Elliot have used multi-variate discrimination function statistics to determine the race from a skull. The discrimination function technique on pelvis, femur, and tibia was developed by **Iscan & Cotton,** and seems to be very objective and easily applied in practice.[2]

Table 4.1 Various Indices.

Race Indices	
Cephalic Index:	$\frac{\text{Max. Breadth of Skull}}{\text{Max. Antero-posterior Length of Skull}} \times 100$
Brachial Index:	$\frac{\text{Length of Radius}}{\text{Length of Humerus}} \times 100$
Crural Index:	$\frac{\text{Length of Tibia}}{\text{Length of Femur}} \times 100$
Inter-membral Index:	$\frac{\text{(Length of Humerus + Length of Radius)}}{\text{(Length of Femur + Length of Tibia)}} \times 100$
Intra-membral Index	$\frac{\text{Length of Humerus}}{\text{Length of Femur}} \times 100$

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Table 4.2 Racial differences from osteometry.

Index	Asiatic Indians	Europeans	Negroes
BRACHIAL INDEX	76.49	75.5	78.5
CRURAL INDEX	86.49	83.3	86.2
INTERMEMBRAL INDEX	67.27	70.4	70.3
INTRAMEMBRAL INDEX	71.11	69.0	72.4

Adapted from **Krogman W M, Iscan M Y.** *The Human Skeleton in Forensic Medicine.* 2nd Ed., **1986**. Charles C. Thomas, USA.

Table 4.3 Racial differences from hair analysis.

Feature	Negroid	Caucasoid	Mongoloid
Diameter	60-90 μm	70-100 μm	90-120 μm
Cross-section	Kidney shaped	Oval	Round
Pigmentation	Dense & clumped towards the periphery	Evenly distributed towards the periphery	Dense auburn throughout the cross-section
Cuticle	–	Medium	Thick
Undulation	Prevalent	Uncommon	Rare

Table 4.4 Sex determination in intersex.

Intersex	Anatomical Sex	Nuclear Sex
Gonadal Agenesis	–	–
Gonadal Dysgenesis Turner's Syndrome Klinefelter's Syndrome	 Female Male	 – Female
True Hermaphroditism	Bisexual	Female
Pseudo-Hermaphroditism Male Female	 Female Male	 Male Female

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Scalp hair also demonstrates characteristic features helpful in the determination of race (Table 4.3)

Certain **dental characteristics** are also helpful in the determination of race. The most distinctive feature is the 'shovel-shaped' incisor seen among American Indians, Chinese, and Palestinian Arab populations.[3] Other features like *cusp of carabelli, protostylid, cusp six & cusp seven, arrangement of groove patterns of molars, accessory ridges on canines, longitudinal tubercles in anterior teeth,* etc., have also been studied for the discrimination of race.[4]

The anterior curvature of the femur is said to be distinctive for the negroid race.[1]

SEX

Though the determination of the sex of a living individual appears to be a simple and straight forward task, it could be problematic in cases of **inter sex** and **concealed sex**.

Sex of an individual can be surmised on the basis of external features like clothing, general contours of the face, distribution of hair, voice, gait, etc., though they are not very reliable. The development of secondary sexual characteristics enhances the probability of reliability, but the most conclusive evidence lies in the presence of ovaries in the female, and the testes in the male.

Sex can also be determined from the presence of *Barr bodies* and *Davidson's bodies* in the nuclei of certain cells.

Barr body or *sex chromatin* (Fig. 4.1) is a distinctive basophilic, intranuclear structure usually located near the inner surface of the nuclear membrane of somatic cells of females, which is absent in males. It is presumed that this is due to the inactivation of an extra X chromosome, and the number of Barr bodies present is correlated with the number of X chromosomes present as follows:

$$B = X - 1$$

(B = number of Barr bodies, X = number of X chromosomes).

This is sometimes helpful in clarifying certain chromosomal aberrations.

A recent study on the frequency of Barr bodies in females of different Asian racial groups produced the following results: Indian (34%), Malaysian (33%), and Chinese (22%).[5]

Davidson and Smith observed a sex difference in the morphology of polymorphonuclear leukocytes

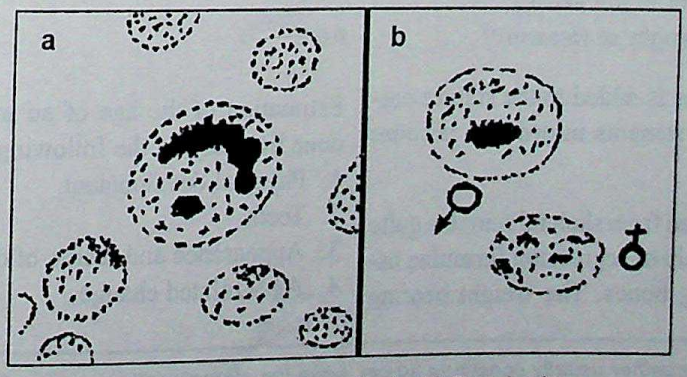

Fig. 4.1 a) Davidson body. b) Barr body.

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in the females. Some polymorph nuclei demonstrate an additional lobe called as a "drumstick" which is rarely found in the males. This has come to be called the "*Davidson body*".

The Y-chromosome present in some cells demonstrates fluorescence when stained with quinacrine.

Table 4.4 mentions some important points in relation to "**Intersex**".

Sexing of skeletal remains can be done on the basis of morphological and morphometric differences of certain bones, and also by multiple discrimination function analysis (**Tables 4.5 and 4.6**)

STATURE

Estimation of stature or height of a person is subject to variations during life due to muscular relaxation and elasticity of intervertebral discs, but could still be valuable in identification.

Stature estimation from dismembered body parts can be done on the basis of the ratio of the body part concerned, in relation to the entire body. Thus

Stature
- = 2 x (length of either arm) + 30 + 4 cms*
- = 2 x (length from vertex to symphysis pubis)
- = 2 x (length from symphysis pubis to either heel)
- = 3.3 x (length from sternal notch to symphysis pubis)
- = 8 x (height of the skull)
- = 5/19 x (length of forearm)**

Generally, 2.5 to 4 cms is added to the stature calculated from skeletal remains in order to account for soft tissues.

Stature can be estimated from skeletal remains quite accurately with the help of regression formulae using the length of long bones. The weight bearing long bones especially of the lower limbs are supposed to provide more reliable estimates of stature as compared to the other bones.

Rollet published statural data estimated by using the length of all the long bones of both the limbs for 50 males & 50 females as early as 1888. Different sets of data were documented for fresh bones as well as dry bones. **Karl Pearson** developed regression formulae based on the data provided by Rollet which became popular in the UK and other parts of the Western world. **Dupertuis & Hadden** suggested using more than one long bone in the calculation of stature, and felt that the tibia and femur provided better estimates than radius and humerus. **Trotter & Gleser** studied the racial differences in stature calculation, and evolved regression formulae for the three major races, as well as the Mexican and Puerto Rican groups [6]. **Steele** devised formulae to estimate stature from fragmented bones [7]. He deduced the ratio of various segments of long bones in relation to their entire length, so that the length of a long bone could first be estimated from its fragments, which can then be applied to regression formulae to arrive at the stature. (**Box 4.1** outlines an Indian case where this was done successfully for the first time).

Pan, Nat and Jit, and **Singh**, have worked out data for the calculation of stature of Asian Indians from the length of long bones.

AGE

Estimation of the age of an individual is usually done by studying the following :

1. Physical development.
2. Teeth.
3. Appearance and fusion of ossification centres.
4. Age- related changes.

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* *The two clavicles together usually constitute 30 cm, while the sternal width corresponds to 4 cm.*

** *Measured from the olecranon to the tip of the middle finger.*

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Table 4.5 Sex differences in the skull and mandible.

Trait	Sex	
	Male	Female
General size	Large	Small
Architecture	Rugged	Smooth
Supraorbital ridges	Medium to large	Small to medium
Mastoid processes	Medium to large	Small to medium
Frontal & parietal eminences	Small	Large
Orbits	Square, lower, smaller with rounded margins	Rounded, higher, larger with sharp margins
Forehead	Steeper, less rounded	Rounded, full, infantile
Cheek Bones	Heavier, more laterally arched	Lighter, more compressed
Mandible	Larger, higher symphysis, broader ascending ramus	Small
Palate	Larger, broader, U shape	Small parabola
Occipital condyles	Large	Small
Teeth	Large, lower M1 often 5 cusped	Small molars, often 4 cusped

Adapted from **Krogman WM, Iscan MY.** The Human Skeleton in Forensic Medicine. 2nd Ed., **1986.** Charles C. Thomas, U.S.A.

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Table 4.6 Sex differences in the pelvis.

Trait	Sex	
	Male	Female
Pelvis as a whole	Massive, rugged, marked muscle sites	Less massive, gracile, smoother
Symphsis	Higher	Lower
Subpubic angle	V - shaped	U - shaped, obtuse angle
Obturator foramen	Large, often ovoid	Small, triangular
Acetabulum	Large, directed laterally	Small, directed antero-laterally
Greater sciatic notch	Smaller, narrower, deeper	Larger, wider, shallower
Ischiopubic rami	Slightly everted	Strongly everted
Sacroiliac joint	Large	Wide
Pre & Post auricular sulcus	Not frequent	More frequent, better developed
Ischial tuberosity	Not everted	Everted
Sacrum	Longer, narrower, evenly distributed curvature, with 5 or more segments	Shorter, broader, marked curvature at S1-2, & S2-5, with 5 segments
Pelvic brim	Heart-shaped	Circular, elliptical
True pelvis, or cavity	Relatively smaller	Oblique, shallow, spacious

Adapted from **Krogman WM, Iscan MY.** *The Human Skeleton in Forensic Medicine.* 2nd Ed., **1986**. Charles C. Thomas, U.S.A

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Table 4.7 Age related changes in foetus.

Months (IUL) / Features	3	4	5	6	7	8	9
Length	9 cm	16 cm	25 cm	30 cm	35 cm	40 cm	45 cm
Weight	30-35 gm	120-130 gm	400 gm	700 gm	1 kg	1.5 kg	2 - 2.25 kg
Nails	present in membranous form	--	upto proximity of tips of fingers	--	upto tips of fingers	--	beyond the tips of fingers
Hair	--	Lanugo	Scalp hair appears	--	Scalp hair 1 cm long	Scalp hair 1.5 cm	Scalp hair 2 cm
Sex	--	differentiable	--	well differentiated	--	--	--
Eyelids	--	--	--	Adherent & eyelashes present	Non adherent & eyelashes present	well formed	--
Brain	--	--	--	Sylvian fissure formed	--	--	--
Intestine	--	Meconium in upper part of small intestine	Meconium upto ascending colon	In transverse colon	in descending pelvic colon	--	in rectum
Scrotum	--	--	--	Empty	--	Corrugated	Occupied by testes
Testes	--	--	--	On psoas muscle	Near the internal ring	In canal (L before R)	In scrotum (L before R)
Centres of Ossification	--	--	Centre for manubruim, 1st segment of sternum & calcaneum	--	Talus & 2nd & 3rd segments of sternum	--	lower end of femur, and cuboid

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Age in the intra-uterine life

The age of a foetus can be assessed by studying the developing morphology, appearance of ossification centres in various bones, and also the germination of teeth (**Table 4.7**).

Haase's Rule:

Haase's rule enables us to calculate the age of a foetus in lunar months from the length of the foetus. Until the foetus reaches the length of 25 cm, the square root of the crown-heel length is taken to arrive at the age. When the length of the foetus is more than 25 cm, it is divided by 5.

Age from birth till 25 years:

a) *Physical features*:
An approximate estimate of the age can be made from the height and weight of a person by referring to standard charts. A study of secondary sexual characteristics can be of added help.

b) *Teeth*:
The development of human dentition in three stages, viz. deciduous dentition, mixed dentition, and permanent dentition, follows a periodic sequence which can be utilised for a more or less accurate estimation of age. Data regarding the eruption and root calcification of deciduous and permanent teeth are mentioned in **Tables 4.8 and 4.9.**

Boyde's method:
Cross striations in the enamel of teeth represent daily incremental lines. The age of an individual can be calculated by counting the number of lines from the neonatal line onwards. This method is applicable mainly to infants. The age so calculated is accurate with an error of around 20 days.[3]

c) *Appearance and fusion of centres of ossification*:
Development and growth of bones take place by various methods of ossification. The process of appearance and union of ossification centres follows a definite time sequence which can be utilised for age estimation. As a general rule, the ageing of bones is more precise with respect to the appearance of centres of ossification than it is with respect to the union of epiphyses. A radiographic method to estimate age from the diaphyseal length of either femur or tibia from 1 to 18 years has been developed by **Hunt and Hatch** who have also studied the annual formation of transverse lines in these bones. **Hoffmann** is of the opinion that the diaphyseal length is a reasonably acceptable means of age estimation for individuals below 12 years, especially in skeletons where epiphyseal and dental data are missing.

Fig. 4.2 displays the ages of appearance and fusion of ossification centres commonly utilised in age estimation.

Estimation of age of an individual over 25 years:

a) *Physical features:*
Features like greying of hair, balding, decrease in stature, changes in the skin, arcus senilis, opacity of the corneal periphery, cataract, etc., give an idea of advancing age.

b) *Changes in the teeth* (**Fig. 4.3**):
After the eruption and calcification of the 3rd molar, (maximum 25 years of age), estimation of age in adults can be done based on the physiological changes of teeth. **Gustafson** used the following criteria[8]:

1. *Attrition* - Wearing down of the incisal or occlusal surface.
2. *Paradentosis* - Loosening of teeth due to gum retraction.

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Table 4.8 Calcification and eruption of deciduous teeth.

Tooth	Calcification	Eruption	Calcification of root completed
Central incisor Lower Upper	5-6 months IUL -- do --	6-8 months 7-9 months	1.5 - 2 years -- do --
Lateral incisor Upper Lower	5-6 months IUL -- do --	7-9 months 10-12 months	1.5 -2 years -- do --
First molar	5-6 months IUL	12-14 months	2 - 2:5 years
Canine	5-6 months IUL	17-18 months	2.5 - 3 years
Second molar	5-6 months IUL	20-30 months	3 years

Table 4.9 Calcification and eruption of permanent teeth.

Tooth	Calcification	Eruption	Calcification of root completed
First molar	At birth	6-7 years	9-10 years
Central incisor	3-4 months	6-8 years	10 years
Lateral incisor	1 year	7-9 years	11 years
First premolar	1.5 years	9-11 years	12-13 years
2nd premolar	2 years	10-12 years	12-14 years
Canine	4-5 months	11-12 years	12-13 years
2nd molar	2.5 - 3 years	12-14 years	14-16 years
3rd molar	8-10 years	17-25 years	22-25 years

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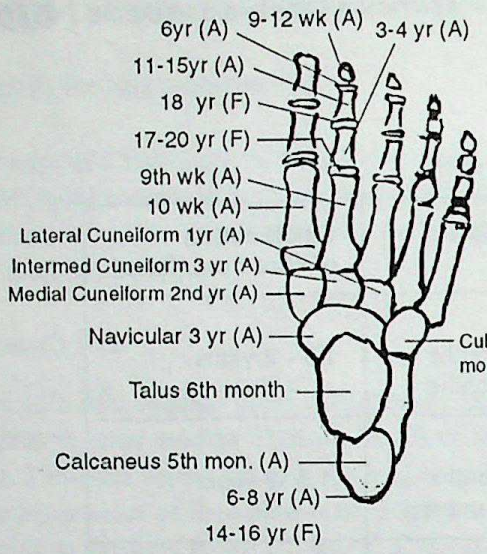
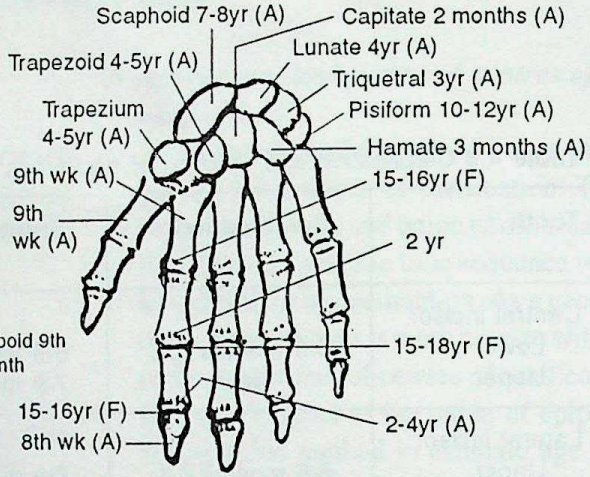
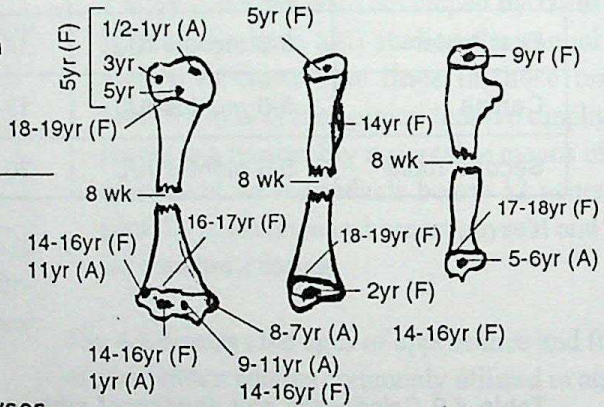


Appearance & fusion of epiphyses in bones of foot



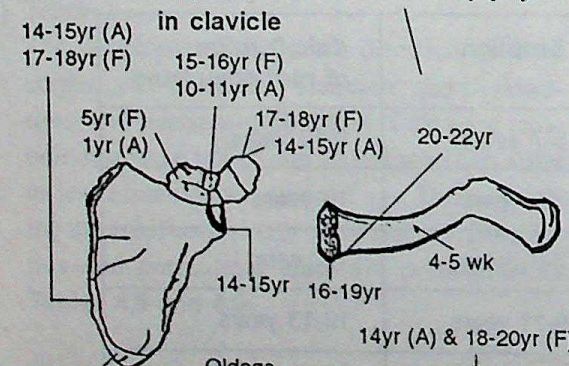
Appearance & fusion of epiphyses in bones of hand

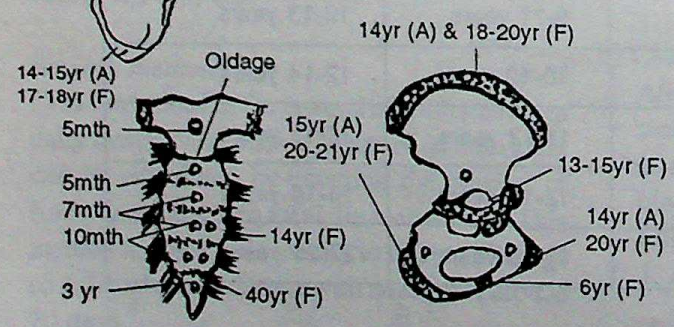
Appearance & fusion of epiphyses in humerus, radius & ulna



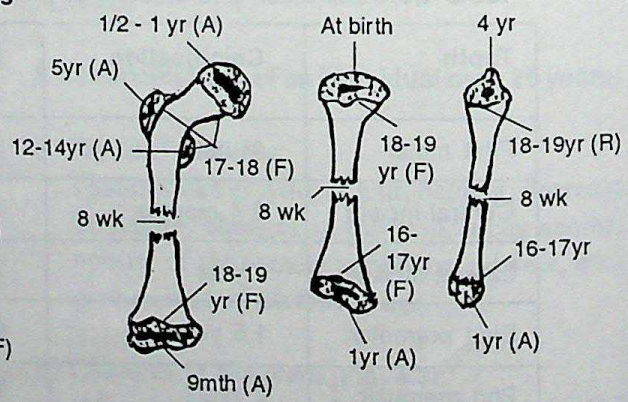
Appearance & fusion of epiphyses in scapula

Appearance & fusion of epiphyses in clavicle





Appearance & fusion of epiphyses in sternum and hip-bone



Appearance & fusion of epiphyses in femur, tibia and fibula

Fig. 4.2

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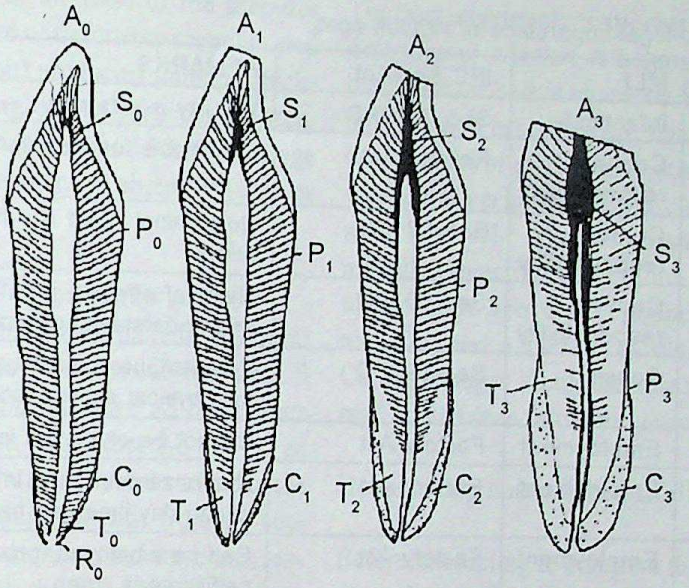

Fig. 4.3 Six changes found in teeth accentuated by age. Taken from **Gustafson G. (1950)**. The changes are A = attrition, S = secondary dentine deposition, P = apical migration of the periodontal membrane attachment, C = cementum apposition, T = root translucency and R = root resorption (not indicated in the illustration).

3. *Secondary dentine* - deposition of dentine in the pulp cavity.
4. *Cementum apposition* - deposition of cementum in the root.
5. *Root resorption.*
6. *Transparency of the root.*

He developed a ranking scale from 0-3 for each of the six criteria. By studying the cross-section of a tooth microscopically, points are assigned to the various changes, and the total score obtained is applied to the following regression formula:

Total score = A + P + S + C + R + T
Age (years) = 11.43 + 4.56 x (total score)
S.E = 3.63

Another method utilises the racemization ratio, (D/L ratio) of aspartic acid to give an accurate estimation of age[9].

c) ***Changes in the skeleton:***

1. *Closure of skull sutures*:
The closure of skull sutures seems to take place from the inner to the outer table, i.e, endocranial to ectocranial surface. Due to the phenomenon of lapsed union, i.e, incomplete union ectocranially, more reliance should be placed on endocranial closure. However, age evaluation by this method is generally not accurate, since it can only provide a range in decades. But it can be considered partially corroborative in the presence of other features.

2. *Age changes in the scapula*:
Stewart summarised the age-related changes in the scapula as follows:
Lipping of the circumferential margin of the glenoid fossa and clavicular facet, appearance of facet on the under side of the acromial process, increasing demarcation of the

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Table 4.10 Medico-legal importance of various ages.

AGE	MLI	IPC Sec/Act	REMARKS
> 7 months IUL	Infanticide	Sec. 300 IPC	Viability is attained
> 5 years	Criminal responsibility	Railway Act	Punishable for the offence
< 7 years	Criminal responsibility	Sec. 82 IPC	No criminal intent & not guilty of the crime
7 - 12 years	Criminal responsibility	Sec. 83 IPC	Guilty of offence, if sufficient maturity and understanding is present
< 12 years	Consent	Sec. 89 IPC	Parents/Guardians must give consent for physical examination.
< 14 years	Employment	Factory Act	Cannot be employed in factory jobs
14 - 15	Employment	Factory Act	Non-hazardous jobs in the factory during day time can be given
> 15 years	Employment	Factory Act	Can be employed, provided a fitness certificate is given
< 15 years (girls)	Rape	Sec. 375 IPC	Sexual intercourse with his own wife, with or without consent.
< 16 years (girls)	Statutory Rape	Sec. 375 IPC	Sexual intercourse with a girl, with or without consent
< 16 years (boys)	Judicial punishment	Juvenile Justice Act 1986	Sent to a reformatory school
< 16 years (boys)	Kidnapping	Sec. 361 IPC	Taking away without the consent of the lawful guardian
< 18 years (girls)	Kidnapping	Sec. 361 IPC	10 years imprisonment, with or without fine
< 18 years (girls)	Kidnapping	Sec.366-A of IPC	Procuring a girl for illicit intercourse 10 years imprisonment, with/without fine.
> 18 years	Consent	Sec. 87 IPC	Can consent to suffer any harm not intended or known to cause death/grievous hurt.
> 18 years	Attainment of majority	Sec.3, Indian Majority Act, 1875	Except those under the guardianship of court Can make a valid will, cast votes in election
> 18 years (girls)	Marriage	Child Marriage Restraint Act, 1978	–
> 18 years	Employment	–	Minimum age to enter government services.
< 21 years (girls)	Kidnapping	Sec. 366-B of IPC	Procuring girls from outside the country or from Jammu & Kashmir for illicit intercourse Punishment 10 years imprisonment with fine.
> 21 years	Attainment of majority	Sec. 3, Indian Majority Act, 1875	For those under guardianship of courts.
> 21 years (boys)	Marriage	Child Marriage Restraint Act, 1978	–
55- 65 years	Employment	–	Age of retirement

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triangular area at the base of the scapular spine, appearance of the cristae scapulae, and atrophy of the bone producing thinning, buckling, and pleating of the infraspinous area. These changes begin around 30 years of age and become prominent at about 50 years or more.[10]

3. *Age changes in the symphysis pubis*:
The formation of a dorsal plateau, a ventral rampart, and the symphyseal rim with 5 stages of development, have all been taken into consideration to predict the age of an individual from the symphyseal surface of the pubis. The age range extends from the second into the sixth decades, and correlates reasonably well with the age changes in long bones and skull.

4. Age can also be studied by a histological examination of long bones in the range between 18 and 85 years.

The medico-legal importance of various ages is outlined in **Table 4.10**

HAIR

The epidermal appendages referred to as hair, constitute a unique characteristic in human beings. Hair is known to resist putrefaction for a long time, and hence attempts were made to determine whether it could have the specificity of fingerprints in identification. Though this has been now proved wrong, a great deal of information can still be obtained from this trace evidence. Before trying to individualise a sample of hair, it is pertinent on the part of the forensic scientist to ascertain *whether it is really hair, or could it be some fibre ?* The distinct morphology of hair with the three layers, viz., cuticle, cortex, and medulla, distinguishes it from fibres which are tubular and devoid of the three layers.

The next step involves **species identification**, i.e., determining whether it belongs to human or animal. *Human hair* is characterised by a thin medullary canal, a broad cortex, medullary index below 0.3, circular or oval cross section, pigmentation towards the periphery of the cortex, and cuticular scale type VII ; whereas *animal hair* has a wider medulla (sometimes filling the entire shaft), medullary index above 0.5, pigmentation around the medulla, and a variety of cuticular scale types. Colour changes along the hair shaft called "banding" is also seen in certain animals.

Human scalp hair can be racially classified.

Before attempting to compare the test hair from the exemplars, and individualise the hair sample, it is necessary to determine the **somatic** origin, i.e, the part of the body from which it originated. Features that can help in pinpointing the body origin of hair include :

a) shape of the tip and the root
b) length
c) diameter
d) contour of cross- section, and
e) character of medulla, cortex, and cuticle.

Sex determination of hair based on observations with reference to the length of hair, and the nature of cut ends, is not reliable. According to **Greenwell et al**, refractive index appears to be one of the best criteria for sex determination, since a significant difference exists between male and female hair. Sex can also be determined by studying Barr bodies in root sheath cells.

An approximate **estimation of age** can be done based on the type of the hair such as primordial, lanugo, terminal, etc, and also from the variation in thickness and distribution of pigments. Various physical factors such as index, size, medullation, cuticular scale count, refractive index, birefringence of the cuticle, and chemical tests such as the rate of

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solution of hair root in caustic potash, etc., have also been studied for the determination of age from birth to maturity.

Further steps in **individualisation** include:

a) ABO blood grouping.
b) Isozyme typing - PGM, RGD, EsD, AK, EAP, GLO, etc.
c) Identification of trace elements by neutron activation analysis.
d) Evidence of disease.
e) Evidence of application of dyes, henna, etc.

Apart from aiding in identification, hair can also help in a variety of ways in medico-legal situations:

1) The type of weapon which caused injury in a particular case can be deduced by examining the tip of the hair. Neatly cut ends suggest the use of a sharp edged weapon, whereas crushed ends result from blunt force injury. Singed hair is characteristic of burns and contact firearm injuries. By examining the root of the hair, it is possible to opine as to whether it fell naturally or was pulled off forcefully during an assault.

2) Heavy metals such as arsenic as well as many drugs of abuse get deposited in the hair, thereby permitting the identification of poison or drug consumed. The deposition of arsenic at intermittent levels on the shaft can also give us useful information regarding the periods of ingestion of the poison. For instance, the analysis of the hair of **Napolean Bonaparte** has proved beyond doubt that he was chronically poisoned with arsenic leading to his death.[11]

HISTORY OF FINGERPRINT IDENTIFICATION[12]

Through the ages there has been a constant quest for a reliable method of identification. This is especially true with regard to identification of criminals, either to link them to their previous criminal history or to a particular crime. **The Prevention of Crime Act** passed in **1871** in Great Britain, and amended in **1877**, culminated in the creation of a **Habitual Criminals Register**, comprising a list of criminals identified by distinctive identification marks such as tattoos, scars from wounds or burns, loss of power of limbs, loss of limbs, disfiguration due to fractures of various body parts, etc. Later this was supplemented by the creation of photograph albums, arranged according to the age and stature of the person and to the type of crime he had committed. Since this method of identifying criminals had only a limited success, a more effective method of identification was felt necessary. Hence the Home Secretary, **Rt. Honorable H.H. Asquith** appointed a committee in **1893** to enquire into a) the method of registering identified habitual criminals then in use in England, b) the anthropometric system prevalent in France, and c) the validity of fingerprint system as suggested by **Galton**.

Anthropometry

In the **Anthropometric system** devised by **Alphonse Bertillon** in **1882** and introduced in France in **1885**, the criminals were classified by certain measurements of specified parts of the body - total height, span of arms, height of trunk, length and width of head, length and width of right ear, lengths of left foot, left middle and ring fingers, left forearm, etc. This method suffered from inherent defects, since cumbersome measurements were required to be made, sometimes leading to error. Also the method was not applicable to children.

Dactylography

Dactylography or **fingerprint system** suggested by **Galton** was based on the accounts of **Henry Faulds & Sir William Herschel** in Nature (**1880**), who claimed that the method was individualistic and constant throughout life. Galton classified fingerprints into three types: arches, loops & whorls.

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But he was unable to sub-classify them which was essential to deal with a collection greater than 2500 persons. **Edward Henry** is credited with a simple classification system which is satisfactory and successful, and is still in vogue since 1900.

Dactylography or the fingerprint system is based on the study of epidermal ridges and their configurations (*dermatoglyphics*) in the palms of the hands. The method can also be extended to the soles of the feet. This method of identification has superseded all the other methods for a variety of reasons:

- Fingerprints are formed during intra-uterine life and remain constant throughout life.
- They are highly individualistic even in monozygotic twins.*
- Fingerprint types can be coded in a form which can be easily filed and retrieved, and also can be teleprinted to various parts of the world.
- They can be compared with latent prints which are developed with suitable reagents, left by the person at the scene of crime. (The credit goes to **Edmund Locard** for the recognition of 'chance' prints)

Today fingerprints are classified into four major groups (Fig. 4.4) :

1. *Arches*
2. *Loops*
3. *Whorls*
4. *Composites*

Arches are further classified as plain or tented, *loops* as radial or ulnar, and *composites* can be either central pocket loops, double loops or accidentals. Individualisation is based not solely on the patterns

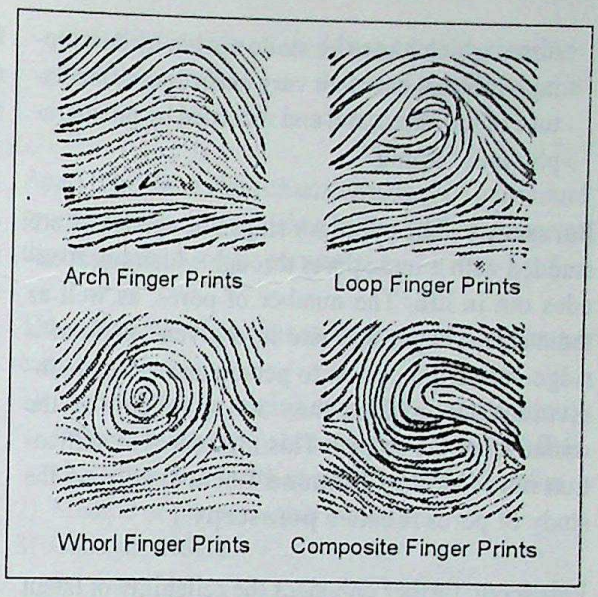

Fig. 4.4 Finger prints.

but extends also to the counting of ridges between the core and the delta, and even the minutiae of ridges like ridge ending, bifurcation, spur formation, dots, lake formation, broken ridge, short ridge, etc. *16 to 20 points of similarity in the minutiae are necessary to establish positive identification.*

Fingerprints encountered at a scene of crime can be classified in the following manner:

1) **Visible print**: impression made by a finger contaminated with blood, paint, dust, dyes etc., which can be recorded by photography with suitable use of lights and filters.
2) **Plastic print**: impression made on a soft substrate like soap, wax, clay, etc., which can be recorded by photography or by means of a cast.
3) **Latent fingerprint**: impression made due to deposition of sebaceous and sweat gland secre-

---

** Studies have shown that fingerprint patterns are inheritable. The patterns and ridge count are similar in monozygotic twins, but can be individualised by the minutiae of the patterns. Though this inherited similarity might pose problems in incomplete, smudged, or vague latent prints, it can sometimes provide information to help adjudicate questions of paternity.[13]*

Dermatoglyphyics has also found applications in clinical medicine, since unusual patterns can be indicative of chromosomal aberrations.[14]

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tions, which has to be made visible by developing with reagents that vary according to the nature of the substrate, and recorded by photography and "lifting".

Poroscopy: The papillary ridges of epidermis are studded with minute pores through which sweat exudes out in life. The number of pores, as well as their size, shape, and site in a given length of a ridge vary from person to person and are quite individualistic. In fact, they are as reliable as the minutiae of the ridges. This method of identification introduced by **Edmund Locard**, utilising the study of pores is called **poroscopy**.

Poroscopy further enhances the reliability of latent fingerprints especially in cases of partial fingerprints where the usual comparison of minutiae cannot be resorted to. Since 9 to 18 pores (per mm) are present in a ridge, and each is unique in its characteristics, this method can lead to positive identification.

FOOTPRINTS

Impressions of human feet on various substrates have been studied and have been found to be quite individualistic with reference to dermatoglyphics, various measurements, and also the morphological analysis.[15]

Dermatoglyphics or the ridge pattern -if clearly visible on the substrate against a contrasting background - is highly individualistic. The comparison of the footprints of the suspect with the test print can be carried out in the same way as in the case of fingerprints. Latent prints are also developed in the same way.

In the absence of ridge patterns, as happens often in substrates such as soil, sand, etc., the measurements like length and width of the foot, length and width of toes, toe pads, the angles of declination from each toe, and from the ball to its juncture with the arch, etc., aid in distinguishing the footprints of one individual from another.

Morphological features such as the shape and size of the contours of each toe, ball, ball-arch juncture, arch, arch-heel juncture, and heel, allow positive identification because of their uniqueness. Even in monozygotic twins, though similarity of features exist, subtle differences occur in the posterior contours of the ball, ball-arch juncture, and arch-heel juncture, which can be utilised for identification purposes.

It has been estimated that stature of an individual constitutes 15 % ratio of foot length, i.e.,

$$\text{Stature} = \frac{\textit{Maximum foot length}}{0.15}$$

The weight of a person can also be estimated by studying the foot outline ball width. It is around 60.5% ratio of the foot outline width in case of males above 14 years, and around 67% ratio in case of females above 14 years.[16]

Moreover, in the case of a walking foot print, i.e., if a series of foot prints are available, it is possible to relate them with the gait pattern of the suspect. The weight distribution, length of the stride, and presence of abnormalities can be studied.

Footprints are recorded by photography or by making casts.

SCARS

A scar is an acquired defect widely accepted as a marker of identification. The size, shape, and location of a scar can aid in positive identification, especially if it is unusual or unsightly.

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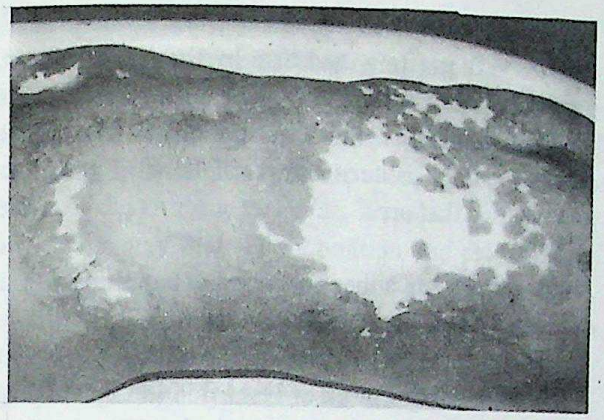

Fig. 4.5 Healed Burn Scar with Hypopigmentation.

A scar is the result of a repair mechanism to an injury involving the dermis of the skin. It is covered by epithelium and is devoid of pigmentation, sweat glands, and hair follicles. Apart from aiding in identification, a scar can also give information regarding (Fig. 4.5)

1. the nature of the injury, and
2. the causative agent.

The size and shape of a scar will be approximately the size and shape of the original wound. Hence the nature of injury, as to whether it is an incised, lacerated, stab wound, etc., can be made out, which in turn can help predict the type of the weapon used to produce the injury.

The age of a scar usually correlates well with the time of infliction of the injury.

CHEILOSCOPY

Cheiloscopy is the study of furrows or grooves present in the human lips. The pattern of the furrows is unique and individualistic, and can be used in some cases of identity fixation.

Cheiloscopic patterns are classified as:

a) *Linear* - Lines are predominant
b) *Bifurcate* - Bifurcation of lines is dominant
c) *Reticular* - Lines cross each other
d) *Undetermined* - None of the features is dominant.

Apart from these class characteristics, 24 individual properties are distinguished in the same manner as the minutiae in the fingerprints.

Three methods of cheiloscopic examination can be employed :

1) Method of determining common features (similar to dactyloscopy): establishment of 7 to 9 fine characteristics leads to positive identification.
2) Method of photographic montage.
3) Contour method.

The second and the third methods supplement each other, wherein the contour of the lips is compared. Apart from this, the substance forming the print, i.e., saliva, cosmetics, etc., can be identified which can act as corroborative evidence.

RUGOSCOPY

Palatal rugae are unique to each individual. The use of human palatal rugae as a method of identification was suggested by **Harrison Allen** as early as 1889. The advantages of palatal rugae as an ideal method of postmortem identification include:

1) They are protected from trauma by their internal location. Also, they are insulated from heat by the tongue and buccal fat pads.
2) No two palates are alike in their configuration. Study of the rugae pattern of twins showed that they are similar, but not identical.
3) Palatoprints do not change during growth.

Thomas & Kotze have classified rugae into 3 types:

Primary Rugae	:	5-10 mm & 10 mm or more
Secondary Rugae	:	3-5 mm
Fragmented Rugae	:	less than 3 mm

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The shape of the rugae could be either curved, wavy, straight, or circular.

TATTOO MARKS

Tattoo marks are designs or patterns imprinted on the skin surface by multiple small puncture wounds with the help of needles dipped in colouring matter.

Tattooing is a form of identification procedure deliberately acquired by a person for various reasons. The practice of tattooing is prevalent among some sections of the people all over the world. (Fig. 4.6)

Fig. 4.6 Tattoo mark

Colouring pigments in common usage include carbon (black), cinnabar, vermilion (red), ochre (brown), chromic oxide(green), prussian blue (blue), etc. The permanence of the mark depends on both the type of the dye used, and the depth of the skin involved. Stable pigments such as carbon, prussian blue, etc., impregnated into the deeper layers of the skin last longer. Even faint/disappeared marks of unstable pigments like cinnabar can be made out on histological study of the regional lymph glands. When tattoo marks are obscured by decomposition or mummification, they can be visualised by treating the area with 3% H_2O_2 which temporarily removes the products of decomposition[19].

Tattoo marks can be removed by surgical methods such as skin grafting, and also by the use of laser beams, without leaving any permanent mark. But crude methods of erasure like application of corrosives, burning, cautery, etc., will leave a permanent scar in that area.

A tattoo mark can either be altered or superimposed over another design subsequently.

Apart from being a mark of identification by virtue of its size, shape, design, anatomical position, etc., tattoo marks can also provide additional information about an individual, viz,

a) Race, religion, nationality: The design prevalent in that particular country/cult may be reproduced.
b) Language.
c) The name tattooed could be that of a person associated/related to him.
d) The mental makeup and sexual preferences of the person can sometimes be made out.
   - for example, the blue bird design on the back of the hand at the base of the thumb and the forefinger, is said to be common among homosexuals.
e) The dates of personal events may be depicted in a tattoo mark.
f) Drug addicts often imprint tattoos over areas where multiple injection marks are present, in order to conceal them.
g) The social status to which the person belongs can be guessed by the nature of the dye used, the design, and also the presence of complications arising out of employing unsterilised needles.

SKULL SUPERIMPOSITION

Skull superimposition is the technique in which the skull to be identified is superimposed on a recent antemortem photograph of the suspected deceased.

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The antemortem photograph is enlarged to "life size", the enlargement factor being based upon the measurements of the fabric on the victim, or other measurable items seen in the photograph, or the focal length of the camera lens used, or the dimensions of the anterior teeth, etc. The skull is adjusted in such a way that the inclination and orientation are the same as that of the head in the photograph. A life size picture is taken and superimposed on the transparency of the life size antemortem photograph, making allowances for soft tissue and hair thickness. The anatomical landmarks compared for a positive match include the following: external auditory meatus, orbits, anterior nasal spine, chin point, angles of the mandible, and zygomatic processes.[20] Arriving at a positive match does not always mean a positive identification, since a photograph of any other person with similar craniometry can produce a successful match with the same skull. Thus, this test can help in exclusion rather than conclusion.

Utilising a methodical, proforma-based approach in superimposition can greatly improve results and minimise errors.[21]

The major disadvantage in photographic superimposition is that a slight change in the enlargement of the antemortem photograph or a slight change in the angulation of the skull from that of the photograph can lead to major discrepancies resulting in a mismatch. Exact determination of the parameters however, results in loss of valuable time. These problems are obviated by the use of video superimposition technique.

Video Superimposition[22]

The skull devoid of any extraneous soft tissue and foreign matter, is mounted on an adjustable support allowing movement in three planes. The antemortem photograph (preferably with the person smiling or displaying his teeth) is also mounted. A colour video camera, firmly mounted on a tripod, is aligned at right angles to the antemortem photograph. The centre of the lens should be at the same level as the horizontal centre of the photograph. The mounted skull is placed in a contrasting background, and a second camera similar to the first one, is aligned to the skull in the same way as the first camera to the photograph. The individual video signals from each camera are fed into a vision mixer so that a variety of functions like horizontal and vertical wiping, superimposition, and negative simulation can be performed.

The skull is oriented on the adjustable mount as closely as possible to the angulation of the head in the photograph. The video pictures from both the cameras are relayed into the video mixer. By adjusting the mixer, the video picture of the antemortem photograph can be enlarged. If teeth are visible in both the skull and photograph, enlargement is carried out till the teeth in the antemortem photograph overlap the teeth in the superimposed picture. If teeth are not present or visible in either specimen, the enlargement factor is estimated by adjusting the vertical facial height of the photograph to the skull. The video mixer offers flexibility in making the adjustments and provides immediate results without having to process photographs or make tracings. After correct enlargements and orientation, superimposition is done, and features are compared with respect to the landmarks. The video mixer allows for the fading in and out of either the skull or the photograph, or for sweeping over each other in the vertical or the horizontal plane. This procedure allows part of the skull or the photograph to be superimposed in either the vertical or horizontal plane on the video screen so that an accurate match is obtained. This technique can be video taped for court purposes.

Note: Superimposition is a technique where a postmortem record is placed over a comparable antemortem record for comparison. This is also used to

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Box 4.1 A Puzzle of Skeletal Remains.

On 30 December 1981, Miss S, a 37 year old woman from a village in Karnataka left home for a nearby town on a personal matter. She never returned. On 28 January 1982, her twin sister Miss SS formally reported the disappearance to the police. On 11 March 1982, policemen discovered decaying bones (part of a skull, and fragments of long bones), along with some hair tufts and feminine apparel in an isolated part of a nearby forest area. These were sent to the Medical Officer of the local Primary Health Centre (PHC) who opined that the bones were of an adult human female, and that they bore signs of antemortem injury. However, he referred the case for expert opinion to the Forensic Medicine Department of the nearby Government Medical College. The report issued subsequently by a specialist from the department contained conflicting information, and therefore the matter was referred through the Corps of Detectives, Bangalore, to the Central Forensic Science Laboratory, Hyderabad, for final opinion.

At the laboratory, Experts in Forensic Medicine and Forensic Science collaborated on the case and systematically set about the task of identity fixation.

Methodology:-

- Precipitin test (*Ouchterlony test*) was done on the hair and bones which established their human origin conclusively.
- All the remains were concluded to belong to the same individual since they had the same configuration, did not demonstrate discrepancies in age or sex, and did not show evidence of duplication.
- Sex was deduced to be female on the basis of feminine characteristics (especially in the skull).
- Age was deduced to be around 35 to 40 years on the basis of suture closure, mandible characteristics, and Gustafson's method. The fact that the dentition indicated only 28 teeth to be present was explained by the (not uncommon) condition of permanent partial anodontia in which the 3rd molars do not erupt at all.
- Height was difficult to determine, since only fragments of long bones were available. However, by utilising specialised formulae (*Steele's formula*), the stature was meticulously worked out to be around 5 feet 1 inch (+/- 1inch). The height of the missing woman had been 5 feet 1 inch.
- Even though the skull was incomplete with some parts missing, reconstruction was accomplished with plasticine, and superimposition done with a recent life size photograph of the missing woman. They matched perfectly.
- An anomalous condition with reference to the mandibular incisors was noticed at the time of examination, which was verified as having been present in the missing woman as per the statements of the surviving sister, other relatives, and friends.
- Some bangles recovered along with the remains demonstrated an average diameter of 5.8 cm which matched perfectly with the average diameter of the bangles belonging to the missing woman (provided by the surviving sister).
- The black dye used in the tuft of hair was analysed as to its constituents and matched with the dye belonging to the missing woman (recovered from her home).
- The cause of death was deduced to be blunt injury to the skull on the basis of antemortem fractures.

Adapted from ***Kashyap VK, Kulashekharan AR, Pillay VV, Patnaik VP. A puzzle of skeletal remains: How forensic science and medicine succeeded in unravelling a complex and controversial murder case.*** Med Sci Law **1990;** 30 : 321-328.

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compare dentition, trabecular patterns, and sinus configuration on x-ray films.

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CHAPTER - 5

DNA Finger Printing

"Your fate does not lie in your destiny, it is locked up in your genes".
- William Fontaine

SYNONYMS

Genetic Fingerprinting
DNA Typing

INTRODUCTION

It has long been the ambition of the forensic scientist to be able to identify the origin of samples of blood, body fluids, or tissues, with the same degree of certainty as to the owner, as *dactylography* (fingerprints). The arrival of **DNA fingerprinting** has revolutionised the concept of identification, and helped realise the ambitions of forensic investigators in achieving pin-point accuracy. Until the advent of this method, a battery of tests had to be done in each case taking into account blood groups, red cell enzymes, serum proteins, and HLA system, all of which display polymorphism to varying extent. However, even if all these systems are utilised in a given case, the chances of inclusion in positive cases will never be 100%. This is because of relatively low degree of variability, as well as, instability of some of these biological markers (especially in old, dried stains or tissues). The quantum of variability is limited because these systems are part of the "expressed" genome, and therefore hypervariability in such genetic sequences is not possible. Too many variations would only lead to failure in expression of the gene altogether, or the production of a defective protein which is nonfunctional. DNA fingerprinting utilises "non-expressed" portions of the genome, which explains the numerous variants for study.[1]

While **Wyman and White** in **1980** discovered the first set of such hypervariable regions (HVRs) in the human genome, today it is known that there are more than 1500 HVRs dispersed throughout the genome. The technique which enables us to study these HVRs which are unique to each individual is called DNA fingerprinting, and was first developed by **Sir Alec Jeffreys** of Leicester University, UK, in **1985**.

PRINCIPLE

The human body consists of 6000 billion cells constituting various tissue and organ systems. Every cell has genetic material contained in units called chromosomes located in the nucleus. There are 23 such pairs of chromosomes, of which 23 are derived from the biological father, and 23 from the mother, as a result of fertilisation of ovum with sperm. Genes are located along the length of each chromosome and are responsible for various functions of the body. Each gene carries instructions for the production of a particular protein which performs a particular function. Genes are also responsible for transmission of heredity.

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All the chromosomes are made up of a very important molecule called deoxyribonucleic acid or DNA, which is sometimes referred to as the chemical blueprint of life. Since chromosomes are confined to the nucleus, it naturally follows that DNA is present only in nucleated cells, though it is true that mitochrondria can also contain DNA. The following points must be borne in mind[2]:

- Each chromosome consists of 2 chromatids joined at the centromere.
- Each chromatid is made of a single DNA molecule.
- Each DNA molecule is made of a double helix which is spiral in nature, and looks in fact like a spiral staircase (**Watson-Crick model**).
- Each strand of this helix is composed of a polynucleotide chain comprising a series of nucleotides joined to each other.
- Each nucleotide is composed of the following: phosphate, deoxyribose sugar, and organic nitrogenous base.
- There are 4 kinds of bases: adenine, guanine, cytosine, and thymine.
- The bases of one strand are connected to the bases of the other strand by means of hydrogen bonds, while adjacent nucleotides are linked with each other by covalent bonds.
- Adenine combines only with thymine, while guanine combines only with cytosine, (and vice-versa).
- Each "step" of the spiral staircase (DNA helix) is formed by a base pair (e.g. A-T, T-A, C-G, or G-C). There are 3 hydrogen bonds between G and C, and 2 bonds between A and T.
- A single DNA molecule comprises 50 million to 500 million base pairs, and is therefore essentially a long, unbranched polynucleotide chain.
- The two strands of the DNA helix have opposing polarity and are said to run anti-parallel to each other.
- The base sequence of one strand is always complementary to the sequence on the other. It provides a code for the synthesis of proteins, and for the storage and transmission of genetic information.
- Each segment of DNA in a chromosome which codes for a particular protein is called a gene. These segments are exactly the same in all individuals. Any variation gives rise to genetic disorders.
- The total number of genes in the human genome is said to be about 10,000. However, suprisingly this accounts for only 2 to 5 % of the entire cellular DNA. The function of the remaining 95% or more of the DNA is not known. Such "non-coding" DNA is referred to as *junk DNA*.
- The *junk DNA* contains regions where some sequences repeat themselves over and over, like a stutter ("repetitive DNA").
  E.g.,...CAGT*GATAGATAGATAGATA*CTGA...
  In the example shown, the sequence GATA is repeated several times and is called "repetitive DNA".
- The regions containing repetitive DNA demostrating hypervariability from individual to individual, are clubbed under the term "Satellite DNA".
- There is an extremely high degree of variability in such tandemly repeated DNA, and these

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variants are called "variable number tandem repeats" (VNTR), or "mini satellites".

- Such VNTR in non-coding DNA is studied in DNA fingerprinting, and helps to conclusively differentiate one individual from another.

- There are more than 1500 VNTRs in the human genome. By breaking up selected regions into fragments with the help of special enzymes (*restriction endonucleases*), such polymorphism can easily be highlighted. The method that is commonly employed for this purpose is called "Restriction Fragment Length Polymorphism" method (RFLP method).

- If the quantity of DNA available for analysis is extremely small, it can be amplified by a technique called "Polymerase Chain Reaction" (PCR).

All these points have been summarised for convenience and presented in **Table 5.1**

PROCEDURE

The commonest method of DNA fingerprinting as devised by Sir Alec Jefrreys, i.e., **RFLP method**, involves the following 7 steps (**Fig 5.1**):

1. **DNA isolation:**
   DNA can be extracted from virtually any body fluid or tissue. The only requirement is that nucleated cells must be present. Material commonly utilised for forensic purposes include blood, bone marrow, semen, tooth pulp, hair root, and organs such as liver, spleen, kidney, skin, etc. Isolation of DNA can be done by the detergent method, phenol method, or the cesium chloride method. Essentially it involves the following steps:
   - Disruption of cells and fractionation of cellular organelles.
   - Dissociation of DNA from proteins by the use of salt solution or detergent
   - Addition of an extractant to phase - separate the bulk of the protein from the DNA.
   - Use of enzymes or differential precipitation to remove the RNA and polysaccharides.

2. **DNA quantitation:**
   The isolated DNA has to be quantitated for study, and this is usually achieved by ultraviolet spectrophotometry.

3. **DNA digestion:**
   The isolated and quantitated DNA is now chopped into fragments with the help of special enzymes called *restriction endonucleases*. These enzymes which work like molecular scissors, cleave the DNA at specific sites, each recognising a particular sequence. There are several restriction endonucleases in use, for e.g., EcoR-I, PsT-I, Hin F-I (all obtained from *Escherichia coli*), Sau 3A-I (obtained from *Staphylococcus aureus*), Hae-III (obtained from *Haemophilus influenzae*), etc.

   A common endonuclease used in forensic practice is PsT-I (a six-base cutter) which recognises the sequence CTGCAG, and cuts the DNA between A and G, wherever it encounters this sequence at a particular locus. The cut fragments will contain VNTR of varying lengths, thereby producing DNA fragments of varying sizes.

   In the USA, the FBI commonly uses Hae III, a four-base cutter which recognises the sequence GGCC.[3]

4. **Electrophoresis:**
   The various DNA fragments are separated on an agarose gel medium using an electrical field. The DNA fragments being negatively charged, move from the negative to the positive end of the field. The larger fragments move slowly and

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Fig. 5.1 DNA finger printing procedure.

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come to a stop near the loading end, while the smaller fragments move faster and migrate to the farther end.

5. **DNA transfer:**
Since agarose gel is extremely fragile and therefore difficult to handle, the separated DNA fragments have to be transferred on to a more resilient medium, for e.g., nylon membrane. This is usually accomplished by a process called *Southern blotting* which utilises capillary action to "suck" the fragments off the gel and transpose them on to the membrane.[4] The result is a mirror-image replica of fragment distribution. Today instead of Southern blotting, *vacuum blotting* technique is employed more commonly, since it consumes much less time.

The transferred DNA fragments are 'fixed' on to the nylon membrane using UV radiation, heat, or air drying.

6. **Hybridisation:**
This step involves the addition of a *probe* to the nylon membrane. A probe is a single-stranded recombinant DNA segment, or synthetic DNA, which is designed to go to a particular predetermined locus on a particular chromosome. It is usually tagged with a radioactive marker, e.g., p^{32}. The probe scans all the DNA fragments and wherever it encounters its complementary sequence, it will hybridize with it, thereby making the fragment radioactive. Normally four probes are used, one at a time in every forensic case, which means that four different regions of the DNA would be analysed at the conclusion of the test. While Lifecodes Corporation (USA) came up with the following probes early on - D2S44, D14S1, DI7S79, and Dzysi4, several other probes have now been evolved. In India, an indigenous probe is commonly used - Bkm, which has been devised by **Dr.Lalji Singh**, one of the pioneers in the field of DNA fingerprinting. It is a multilocus probe, and consists of a sex chromosome - associated repetitive DNA isolated from the female banded krait (a venomous snake), as a minor satellite DNA. More than thirty six hypervariable VNTR are detected by Bkm. A full fledged **Centre for DNA Fingerprinting and Diagnostics** has been established by the Government of India at Hyderabad, Andhra Pradesh.

7. **Autoradiography:**
Once the probe binds to the nylon membrane, it is invisible to the naked eye and must be visualised. This is accomplished by a technique called *autoradiography*.

First, the excess probe is washed away from the nylon membrane. The membrane is then placed against an x-ray film and exposed for 1 to 10 days. When the film is subsequently processed, grey to black bands appear where the radioactive probes had hybridised to fragments bearing complementary sequences. The series of bands seen on the film is called an "autorad", and essentially represents the DNA fingerprint of that individual from whom the DNA had been obtained (Fig 5.2).

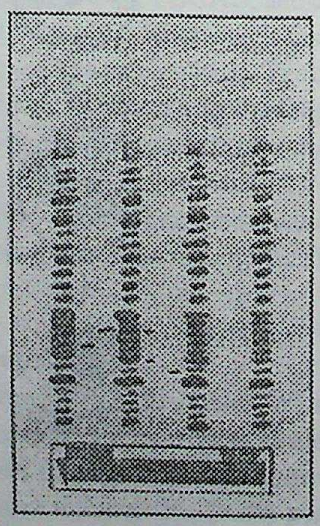

Fig. 5.2 Autorad

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The pattern of bands is unique to each individual, and the probability that two individuals could have the same pattern is said to be one in several billions.

Approximate time taken for each step of DNA fingerprinting (RFLP method):-

1. DNA isolation	:	few hours to 3 days.
2. DNA quantitation	:	1/2 hour
3. DNA digestion	:	8 to 12 hours
4. Electrophoresis	:	12 to 16 hours
5. DNA transfer	:	
Southern blotting	:	1 to 2 days
Vacuum blotting	:	5 to 6 hours
6. Hybridisation	:	Several hours
7. Autoradiography *	:	1 to 10 days

Polymerase Chain Reaction (PCR)

Since most forensic samples contain only minute amounts of DNA, PCR is a very valuable tool for the forensic scientist as it is capable of copying the available DNA a million-fold times.[5] The following steps are involved:

1. **DNA isolation:**
   Described under RFLP method.

2. **Amplification:**
   It is the crux of PCR and involves the following steps:
   - *Denaturation* - This means that the DNA is split apart, and is usually accomplished by heating the sample to more than 94° C for a prescribed period of time.
   - *Annealing* - This step requires lowering the temperature, during which small segments of DNA (called "primers") bind specifically to the DNA which has the polymorphic regions of interest. The primers act as the starting point for the DNA duplication process.
   - *Extension* - The temperature is raised to 72° C, which causes the enzyme, a DNA polymerase (*TAQ polymerase*) to extend the primers and copy the two separated strands of DNA. The TAQ polymerase utilises nucleotides by inserting them opposite the appropriate base, thereby ensuring the integrity of DNA's base pair complementarity. Once the primers have been extended and the DNA copied, the cycle is begun again. A run of 30 or more cycles will permit more than a million copies of DNA to be synthesised.

3. **Detection:**
   Detection of the amplification products for VNTR polymorphisms is usually accomplished by electrophoresis, followed by visualisation by fluorescent detection or silver staining.

   Sequence polymorphisms, such as for HLA - DQA1, the common detection method is to use a reverse clot blot procedure, and the amplification products are labelled with a chemical called biotin. After the amplification products bind to a strip containing allele- or sequence-specific probes, a reaction using horseradish peroxidase and a coloured dye causes blue dots to appear. The pattern of blue dots denotes which alleles are present.

APPLICATIONS

1. Rape:
   Seminal fluid or stain from the body or clothing of a rape victim can be matched with blood sample of the accused. DNA fingerprinting enables to implicate a rapist with pin-point accuracy,

---

** A new technique called **chemiluminescence** which utilises enzyme-labelling instead of radioactivity, is much faster and takes only a few hours for processing.*

Box 5.1 details one of the earliest and most sensational cases where DNA fingerprinting helped to exclude thousands of innocent men, while fixing the sole culprit, enabling his conviction and punishment. Incidentally, Sir Alec Jeffreys was directly involved in this case which is today considered a landmark in the field of criminal investigation.

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while at the same time helping to exclude or absolve innocent suspects.

2. Murder:

   a) Blood stains on the clothing or person of the accused involved in a "knifing murder" can be matched with the blood of the victim.

   b) Blood or hair roots on a weapon used to murder a victim can be matched with that of the victim.

3. Identification of mutilated, dismembered, or burnt bodies:

   The DNA fingerprint obtained from such remains can be compared with that of close blood-relatives of the deceased. Since DNA is inherited, links between family members can be established.

   Box 5.2 Illustrates one such case.

4. Paternity dispute:

   Every band in the DNA fingerprint of a child must originate either from the father or from the mother, i.e., half the number from each parent.

   In resolving a paternity dispute, first the bands in the DNA fingerprint of the child are matched with the bands of the mother. Naturally, the remaining bands must match with the pattern of the "disputed" father. If they do correspond, he is the "genuine" father, and if they do not, he is not the father of the child.

COLLECTION and PRESERVATION OF SAMPLES[6]

Medical officers and forensic pathologists must know the nature of material required to be submitted for DNA analysis in a given case, as well as the method of preservation and despatch.

Table 5.2 is a summary of necessary information in this regard.

ADVANTAGES

1. It is the most conclusive test available in the field of crime investigation
2. Since DNA is very stable, the test can be done even on very old (aged) stains or specimens.
3. Very little blood or tissue is required for the test.

DISADVANTAGES

1. DNA fingerprinting cannot differentiate between identical twins.
2. It is a very expensive test.
3. It is a time consuming exercise and normally requires several days or weeks.
4. Interpretation of results requires a great deal of expertise and experience.

Table 5.1 Principles of DNA Fingerprinting

- The "expressed" part of the human genome accounts for only 5 to 10% of the DNA.
- More than 90% of human DNA is not expressed (non-coding), and is referred to as "Junk DNA".
- Junk DNA has several regions where base sequences are repeated tandemly (VNTR)
- VNTR shows tremendous variablility in different individuals.
- The study of such hypervariable regions (HVRs) is referred to as DNA fingerprinting.
- The commonest method used is RFLP (Restriction Fragment Length Polymorphism).
- If the quantity of DNA available is too small, it can be amplified and studied by PCR (Polymerase Chain Reaction).

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Table 5.2 Collection and Preservation of Biological Samples for DNA Fingerprinting.

	Blood		Semen		Urine		Saliva		Tissue	
	Liquid	Stain	Liquid	Stain	Liquid	Stain	Liquid	Stain	Fresh	Dry
Minimum Quantity	1-100 µl or few drops	Size of a 50 paise coin	1-20 µL or 1-2 drops	As much as available	60 to 100 ml	As much as available	Few drops (containing cells)	As much as available	100 to 500 mg	Portion likely to contain viable cells
Preservation	EDTA tube, Refrigerate	Dry, Refrigerate	EDTA tube, Refrigerate	Dry, Refrigerate or freeze	Freeze	Dry, freeze	Dry, freeze	Dry, Refrigerate or freeze	Freeze	Dry, freeze

Adapted from **Geberth VJ.** *Practical Homicide Investigation.* 3rd edn, **1996.** CRC Press, USA. p.563

Box 5.1

The Blooding

At 7 pm on Nov. 21, 1983, 15-year-old Lynda Mann of the British midland village of Narborough left her home to visit a girlfriend. She never returned home. The following morning her body was discovered, the victim of a savage rape and strangulation, near a footpath named the Black Pad which abutted the grounds of the Carlton Hayes mental health hospital. The first murder investigation in the history of the quaint village of Narborough convened, led by Inspector Derek Pearce with more than 150 officers at his disposal. Every male became a suspect to the petrified community, even the victim's stepfather who acquitted himself only after a blood sample failed to match a trace of semen taken from the victim. The high sperm count indicated the assailant to be a male between the ages of thirteen and thirty-four, and with the use of computers, police compiled a list of suspects.

But they failed to find the culprit, and by April 1984 the intense investigation of 150 police men had dwindled to a nominal investigation by only eight men. In August the investigation was curtailed completely, after blood tests administered to 150 suspects had failed to unveil the killer.

Less than ten miles from the murder scene, Alec Jeffreys, a 34-year-old geneticist at Leicester University, was probing the evolution of human genes through their genetic material (DNA). The research was elusive as Jeffreys discovered a variance in the DNA from each human sample. In September 1984, Jeffreys discovered that by adding enzymes to fragments of blood cells and separating larger fragments from smaller by exposing them to an electric field, the resultant blotting onto a nylon membrane would reveal a radioactive pattern similar to a supermarket bar-code, that was unique to every individual. Further, he learned that the bands of genetic fingerprints were inherited from both parents, and that the bands were consistent in any bodily material. In March 1985, Jeffreys stated that the possibility of two people having the same genetic fingerprint was literally none.

On July 31, 1986, 15-year-old Dawn Ashworth of Enderby, England, was seen alive for the last time at 5 p.m. as she entered the Ten Pound Lane footpath which connected neighboring Narborough. On the morning of Aug. 2, police discovered the girl's body under heavy brush at the side of the path. She

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had suffered a vicious beating after being raped, and was then strangled to death. On Aug. 8, 1986, police arrested 17 year old Richard Buckland, a kitchen worker at the mental health hospital. After intense interrogation the simple-minded youth confessed to the crime. But at the suggestion of Buckland's father, samples of semen found in both victims were sent to Dr. Jeffreys.

The police were shocked when Jeffreys stated that Buckland could not have been the murderer. On Nov. 21, 1986, on the date his murder trial was to begin, Richard Buckland became the first suspect in history to be released because of evidence refuted by genetic fingerprinting. A squad of fifty officers was assembled to reopen the investigation, headed by Inspector Pearce.

On Jan. 17, 1987, the police announced that all males between seventeen and thirty-four years of age would be required to provide blood and saliva samples. It proved unwieldy as the men were not required by law to provide samples, and those who did submit could easily circumvent the identity process by providing false ID's, as British drivers licenses did not include a photograph. And dozens of the potential perpetrators were incarcerated in a mental hospital. Even though 90 percent of the men readily responded, the laboratory was overtaxed to test the samples.

By May, more than 3,500 men had submitted to the testing, but only half the samples had been tested. One thousand more needed to be tested, but the manpower had been cut to twenty-four officers and soon to only sixteen. Yet the testing continued, often requiring one of the few remaining men to travel hundreds of miles for a single sample. Six months after the second murder, virtually every potential perpetrator in the area had been tested, but no sample matched the indelible evidence left by the killer of the two teenaged girls.

On Aug. 1, 1987, while drinking in a pub, a bakery worker named Ian Kelly casually mentioned to a co-worker that he had taken the blood test for a fellow worker named Colin Pitchfork. The following day Ian Kelly was arrested for conspiracy to pervert the course of justice. Kelly stated that Pitchfork had begged him to take the test as he had done the same for another co-worker who had a history of sexual perversion. That same afternoon, Aug. 2, 1987, 27-year-old Colin Pitchfork was arrested on suspicion of murder shortly after he returned to his home. Pitchfork, married and the father of two, was a professional cake decorator who also had a history of sexual perversion. He was a compulsive "flasher" and was also known as a molester of female employees at the bakery. Before arriving at the police station, he had confessed to the two murders. On Sept. 21, Pitchfork was remanded at the Castle Court in Leicester on two counts of murder.

The murder trial began on Jan. 22, 1988, at the Castle Court in Leicester before Justice Otton and ended the same afternoon. Ian Kelly was given a suspended eighteen-months sentence for his unwitting cooperation in the cover-up. Pitchfork confessed to both murders and indecent assault charges, as well as conspiracy for involving Ian Kelly. He was sentenced to life for the two murders, ten years for both rapes, three years for both sexual assaults, and three years for conspiracy. The sentences were to be served concurrently, but the judge refused to name a minimum term which meant Pitchfork might be eligible for parole after serving a mere ten years. As life returned to normal in the small villages, the conclusiveness of DNA fingerprinting soothed the usual gnawing doubts after a murder conviction. Many encouraged the reinstitution of the death penalty after seeing how the genetic fingerprint of Colin Pitchfork perfectly matched the grisly evidence that he had left with Lynda Mann and Dawn Ashworth.

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Box 5.2

DNA from Cremains

In December 1987, the police department of Wichita, Kansas, USA, received a complaint from a caller who claimed that a bucket present in the garage of a neighbour's home contained parts of a human body. The police rushed to the scene and recovered the bucket which did indeed contain burnt remains of a human being comprising fragments of bone and burnt flesh. The resident of the home from where the bucket was retrieved was arrested. Subsequent investigation suggested that he had killed his wife who had separated from him, and attempted to cremate her in a funeral home where he worked as an usher. Since one of the burners of the electrical crematorium in the funeral home malfunctioned, the body had been only partially burnt. The murderer had then removed the remaining cremated body (cremains), stuffed them into a bucket, and taken home for later burial in an isolated spot.

In order to prove the identity of the cremains beyond doubt, a fragment of a femur and some tissue scrapings were sent along with blood samples of the victim's parents, for DNA analysis to the Lifecodes Corporation, an accredited private laboratory. Lifecodes extracted DNA from the cremains and positively established the identity by comparing the bands with the bands present in the DNA fingerprints of the parents. Conviction of the accused resulted, who had erroneously believed he had committed the perfect crime ("No body, no crime").

Courtesy **Chief John Dotson**, Sparks Police Department, Nevada, USA.

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CHAPTER - 6

Thanatology

"In the final analysis, it is our concept of death which decides our answers to all the questions of life".

- Dag Hammarskjold

PART ONE

DEATH AND ITS MEDICOLEGAL IMPORTANCE

Thanatology is a branch of science that deals with the study of death. Human life, in a manner of speaking is a brief spell of existence between two heartbeats, man's first and man's last. Death is an inevitable reality and in the past was considered to be a simple and straightforward phenomenon. The doctor would declare a person dead once he was convinced that there was cessation of life in the patient. This meant that the patient had stopped breathing, his heart had stopped beating, there was unresponsiveness, and his body had turned cold. But this state can also exist, though for a brief period, when a person is in a state of *suspended animation* as seen in the victims of electrocution, drowning, hypothermia, etc. Though cessation of heartbeat was enough to consider a person as dead in the past, it is no longer an evidence of death today since the heart is now able to be substituted with that of a ' just deceased donor' or a mechanical one. Moreover, modern biomedical innovations like the resuscitator and cardiac pacemaker have made it imperative to establish a set criteria by which the moment of death could be identified. The basic concept is that death is the irreversible loss of the capacity for consciousness and capacity to breathe. Since both the functions reside in the brain stem, the modern concept of diagnosing death is to identify whether a person's brain stem is alive or not.

BRAINSTEM DEATH

What defines a human being as an independent biological unit is related to a few cubic centimeters of tissue in the floor of the aqueduct between the third and fourth ventricles of the brain. If this small area is dead the person becomes irreversibly unconscious and irreversibly apnoeic.[1]

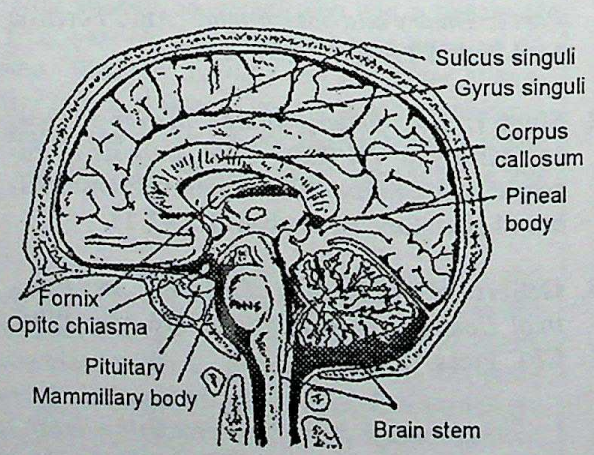

Fig 6.1 Location of brainstem.

The diagnosis of brainstem death:
The diagnosis of brainstem death involves three steps:

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a) The patient is comatose and unable to breathe spontaneously, and therefore unconscious and on a ventilator. The cause of coma must be 'irremediable structural brain damage'.
b) Exclusion of the cause of coma as due to drugs, hypothermia, or profound metabolic disturbance which may reversibly impair brainstem function, and
c) Establishing that all the brainstem reflexes are absent and that the patient fails to breathe, even when taken off the respirator, after certain preliminary changes made in the blood gas concentration (*apnoeic test*).

Testing the brainstem reflexes[2]

- Pupillary response to light (afferent 2nd and efferent 3rd cranial nerve) - making the pupil constrict.
- Corneal reflex (afferent 5th and efferent 7th cranial nerve) – blinking response to corneal stimulation.
- Vestibulo-ocular reflex (afferent 8th and efferent 3rd & 5th cranial nerves) - Dolls eye movement in response to irrigation of the tympanic membranes of the ears with ice- cold water.
- Grimace (afferent 5th and efferent 7th cranial nerve) - in response to painful stimulus over face, and
- Gag or cough reflex (afferent 9th and efferent 10th) - in response to intubation.

If all the above mentioned reflexes are tested properly and are found to be absent, it means that one is examining the brainstem slice by slice, in a series of tests whose results reinforce one another in confirming the fact that the brainstem is dead.

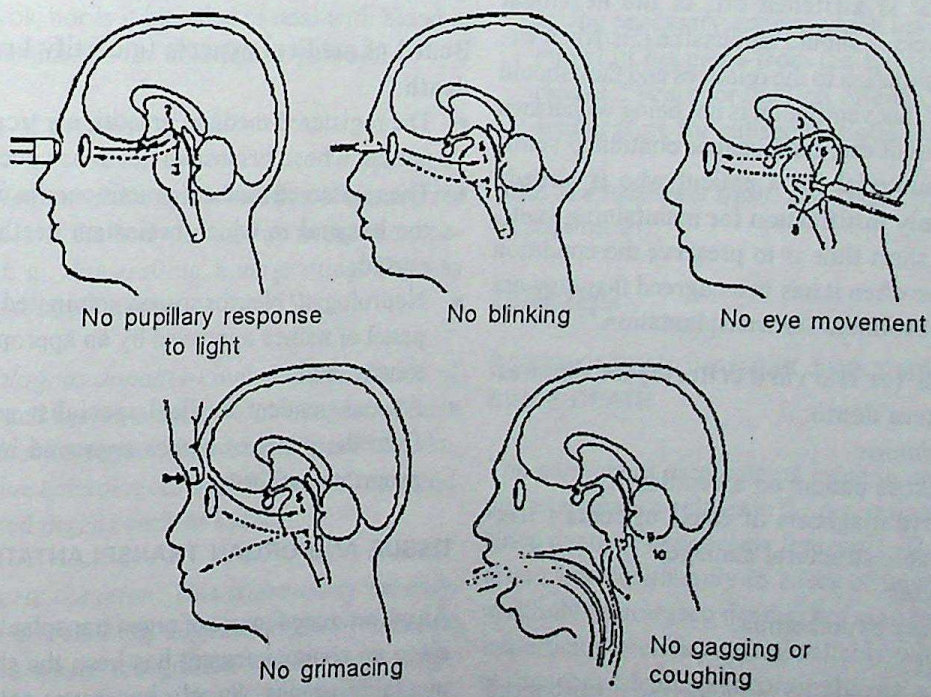

Fig. 6.2 Brainstem reflexes.
Adapted from **Pallis C.** *Brainstem death - The evolution of a concept.* Medico-Legal Journal **1987**.

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The **apnoea test** aims at proving that the patient is incapable of breathing. In doing this test one disconnects the patient from the ventilator, and allows the carbon dioxide to build up to a point where it would trigger off respiration if there were any cells still alive in the respiratory center of the brainstem. The ventilator is disconnected for a period of ten minutes, but to prevent anoxia developing, one must pre-oxygenate the patient thoroughly before the test. This is done by administering 100% oxygen for 10 minutes, then making the subject breathe a mixture of 95% oxygen and 5% carbon dioxide which ensures that one starts with high oxygen and reasonably high carbon dioxide. At the end of the disconnection period, one should estimate the carbon dioxide in the arterial blood to ensure it has reached a level of 50 mm Hg.

The time of death is the time at which *brainstem death* is established, and not some later time when the ventilator is switched off, or the heartbeat ceases. Clinicians should emphasize this when explaining the situation to the relatives and they should make it clear that ventilation is not being withdrawn to let the patient die, but because continued ventilation is immaterial for a patient who is already dead. The only justification for maintaining ventilation for a short time is to preserve the condition of the organs when it has been agreed that they are to be made available for transplantation.[3]

British code (or Harvard criteria) for diagnosing brainstem death:

1. *Preconditions*
   - Comatose patient on a ventilator.
   - Positive diagnosis of cause of coma (irremediable structural damage).
2. *Exclusions*
   - Primary hypothermia.
   - Drugs.
   - Severe metabolic or endocrine disturbances.
3. *Tests*
   - Absent brainstem reflexes.
   - Apnoea.

Although the heart may have stopped, the respiration has ceased, and the brainstem is functionless, tissues retain the 'vital principle of life' for some time. This is known as *somatic death*. The body is dead as a whole, but the biological properties of the component parts may still be demonstrable. The muscles contract to mechanical or electric stimulus. The pupils respond to appropriate pharmacological stimuli. But the time during which these properties persist soon runs out. When these individual tissues and cells die it is termed *molecular death*. The whole body is now dead. In this way, a person dies in bits and pieces. Therefore death is a process, not an event.

The legal certification of death depends on the diagnosis of somatic death. Organ transplantation depends on the persistence of the properties of living matter in the component parts before we die in bits and pieces, i.e., before molecular death sets in.

Board of medical experts to certify brainstem death:

- The registered medical practitioner treating the patient whose brainstem death has occurred.
- The registered medical practitioner in charge of the hospital in which brainstem death has occurred.
- Neurologist/ Neurosurgeon nominated from the panel of names approved by an appropriate authority, and
- An independent medical specialist nominated from the panel of names approved by the appropriate authority.

TISSUE AND ORGAN TRANSPLANTATION

An unfortunate feature of organ transplantation ever since its commencement has been the shortage of available organs. Supply has never satisfied demand, and this has led to the continuous development of procedures and systems to increase supply. Rational argument can be made to the effect that

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shortage has led to the rise of commercial traffic in human organs, particularly from living donors who are unrelated to recipients.

On May 13, 1991, the World Health Assembly endorsed a series of guiding principles on organ transplantation.[4] These guiding principles are intended to provide an orderly, ethical, and acceptable framework for regulating the acquisition and transplantation of human organs for therapeutic purposes. In India, the **Transplantation of Human Organs Act 1994** came into force in 1995 for the regulation of removal, storage and transplantation of human organs for therapeutic purpose and for the prevention of commercial dealings in human organs.[5]

The term *human organ* is understood to include organs and tissues not related to human reproduction, and accordingly does not extend to reproductive tissue- namely, ova, sperm, ovaries, testicles or embryos, nor is it intended to deal with blood or blood constituents for transfusion purpose.

Sources of organs

a) *Homologous donation*- relocation of tissue from one part of the body to another in the same patient. E.g., skin grafting, bone grafting. This has no legal implications.

b) *Heterologous donation*- includes transfusion of blood and bone marrow, which because of their regenerative capacity, pose no threat to the donor. Live heterologous donation can also extend to paired organs such as kidney.

c) *Cadaveric donation*: This is obviously the only means of obtaining unpaired organs[6]. This is the area where recognition and accurate diagnosis of brainstem death acquires great importance, because organs taken out from 'beating donors' have more chances of successful grafting.

Organs from dead bodies can be removed if the donor had left a witnessed authorization. In the case of minors, one of the parents can authorize.

For live unrelated kidney donation, approval by the relevant state authorization committee (*Appropriate Authority*) is necessary. The sale of organs from the dead or living is absolutely illegal. Punishment for conducting, associating with, or helping in removal of human organs without authority will be imprisonment upto 5 years, and fine upto Rs 10,000/-. Whosoever engages in commercial dealings in human organs will be punished with imprisonment for a term varying from 2 to 7 years, and fine ranging from Rs. 10,000/- to Rs 20,000/-. When a registered medical practitioner is convicted under the Act, the Appropriate Authority will report the name to the State Medical Council for taking necessary action, which may include removal of his name from the Register for a period of 2 years for the first offence, and permanent erasure for subsequent offence.

(Also see Brainstem Death and Organ Transplantation on page no. 21).

PART TWO

POSTMORTEM CHANGES AND TIME SINCE DEATH

The paramount medicolegal issue in any postmortem examination relates to the determination of time since death (*Postmortem Interval*). This question arises most commonly in cases of unwitnessed or unreliably witnessed deaths, but may be of crucial importance even in cases of reliably witnessed 'simultaneous' deaths. Accordingly, the accurate determination of the postmortem interval applies not only to civil law, in which ascertaining the exact time of death is of practical necessity in settling

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family, social, and business matters, but also to criminal law, wherein the accurate determination of the time since death may either exonerate or inculpate a suspect accused of a particular homicide. Such determination is an indispensable component of *corpus delicti* (i.e., the body of evidence which proves the commission of a crime), and may serve as a basis for deciding whether to extend investigative effort in a particular line or direction. Therefore it is extremely important to accurately interpret the changes that occur after death.

For convenience, these changes are being described under the following headings:

Immediate changes which result in complete and irreversible cessation of the functions of the brain, heart and the lungs. Muscles of the body become flaccid, and the corneal reflexes are abolished.

Early changes which take place between the first 12 to 24 hours. They comprise

- Changes in the skin
- Changes in the eye
- Algor mortis
- Livor mortis
- Rigor mortis

Late changes include putrefaction and its modifications, namely adipocere formation and mummification.

The immediate changes after death mentioned above may also be seen in cases of **suspended animation**. Suspended animation is a state where the metabolic needs of the body are at such low ebb that the person appears apparently dead. His pulse is not palpable, heart sounds are not audible, respiratory movements are not visually perceptible, and reflexes are either absent or not possible to elicit. The person is not actually dead, and he can be revived in most cases by resuscitation. Recognizing this condition is important in order to prevent premature certification of death. Newborn babies and victims of electrocution, drowning, hypothermia and sedative poisoning may go into a state of suspended animation.

EARLY CHANGES

Changes in the skin: Elasticity is lost soon after death. Skin that was translucent in life becomes pale.

Changes in the eye:

a) The cornea loses its lustre with the passage of time. It becomes dull, hazy, and finally opaque and wrinkled. If there is a *living will* for the removal of the cornea for transplantation, it should be removed preferably within half an hour after death.
b) Intra ocular tension falls so rapidly that by two hours after death, the tension is nearly zero and the eyeball appears sunken.
c) If the retina could be viewed through an ophthalmoscope, the columns of blood in the vessels may be seen to be broken up into segments, which is referred to as 'trucking'.

Algor mortis (Postmortem cooling; Chill of death): There is a constant balance between heat production and heat loss during life. After death, heat production is lost and therefore the body starts cooling. The dead body loses its heat by conduction, convection and radiation. Measurement of the rate of cooling helps in estimating the time since death. When the rate of cooling is plotted on a graph the pattern of the curve assumes a sigmoid shape.**(Fig. 6.3)**

The body surface starts losing heat rapidly, but the core body temperature does not alter until a gradient is established between the core body temperature and the surface. As there is no significant change in the core body temperature for sometime which may be upto 2 to 3 hours after death, an initial plateau is seen. When once the gradient is established between the core body temperature and

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the surface, the rate of cooling is approximately proportional to the difference in temperature between the body and the surface. This aspect is in accordance with the Newton's law of cooling. That means after the initial plateau the curve dips down progressively before it becomes nearly flat at the bottom. The curve may even rise before becoming flat due to heat produced by putrefactive changes.

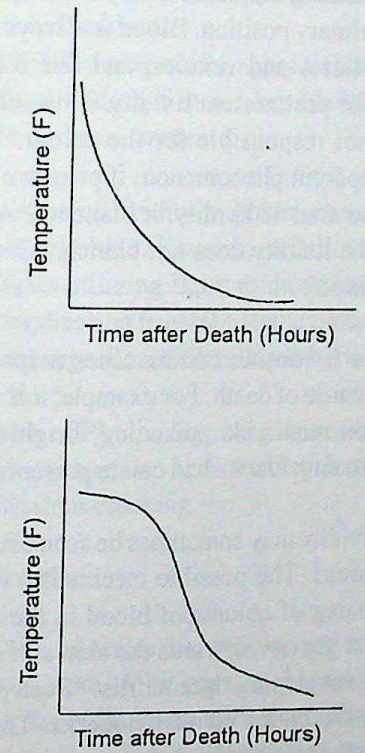

Fig 6.3 Graphs showing fall in temperature when (a) an inorganic body cools, and (b) a dead body cools.
Adapted from **Gordon & Shapiro**, *Forensic Medicine*, **1982.**

The preferred site for measuring the inner core body temperature is either the rectum or the abdominal cavity. A chemical thermometer with graduations from 0 to 50° Celsius is introduced deep inside the rectum (to about 10 cms), or alternatively, the thermometer may be kept in contact with the undersurface of the liver through a slit made in the anterior abdominal wall in the right hypochondrium. At the same time, the environmental temperature is also measured. The time since death is then calculated (after taking two readings at an interval of one hour), by the following formula :

$$\frac{\text{Normal body temp.} - \text{Rectal temperature}}{\text{Rate of fall of temperature per hour}} = \text{Time since death}$$

There are a number of variables :

a) The body temperature at the time of death is presumed to be normal. This may not be the case. In fact in some cases, there is a slight rise in the temperature of the body after death. This is known as **postmortem caloricity**, and may be seen in bodies which at the time of death had suffered violent muscular contractions, or in the case of asphyxial deaths such as strangulation, or septicaemic deaths, strychnine poisoning, pontine haemorrhage, etc.
b) If the environmental temperature is extremely cold, the process of cooling of the body is hastened, while in very hot climates the body may not cool at all.
c) Obesity lessens the heat loss, as fat is a bad conductor of heat.
d) Ratio between the body surface and the body mass – children and old people cool more rapidly.
e) Presence of thick clothing at the time of death slows the rate of heat loss.

In addition to the commonly employed sites for measuring the body temperature mentioned above, other sites have been proposed by various investigators, including the trachea, nasal cavity, middle ear cavity, etc.[7,8] Electronic thermometer and thermocouple have been employed in the place of chemical thermometer by some investigators.[9]

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Livor mortis (Postmortem lividity; Postmortem staining; Hypostasis):

With the stoppage of functions of the heart after death, active circulation of blood in the body ceases. The blood which is fluid in nature, gradually settles down in the toneless capillaries of the dependent parts of the body, seen externally as patches of purplish discolouration through the skin. This settling of blood in the dependent parts is in accordance with the law of gravity. Thus if the body is in supine position the postmortem lividity will be seen on the back. In deaths due to hanging, lividity is seen on the hands, feet, and the lower parts of the face above the ligature. In drowning where the body usually floats with the back facing up and the limbs hanging down, the lividity is seen over the limbs, abdomen and the face. Distribution of the lividity thus is useful in indicating the position of the body after death.

Those parts of the body which are in actual contact with the surface they lie on, do not show the colour as the toneless capillaries are pressed by the weight of the body. Such areas are known as areas of *contact flattening*. Thus in the body which is lying supine, postmortem lividity is not seen over the shoulder blades, buttocks, calves, etc. If the deceased was wearing a tight collared shirt, a band of pale area may be seen around the neck where lividity does not develop, and this could be confused with a strangulation mark by an inexperienced person. Proper interpretation of such findings is important.

Postmortem lividity begins as mottled patches ranging from 1 to 2 cms in diameter. These patches gradually increase in size with the passage of time and coalesce with each other till they become one uniform area of staining. Though the time of the appearance of these patches is variable, they are generally seen 2 to 3 hours after death. If the position of the body is altered, these patches gradually disappear, and appear in the new dependent part. If the body is left undisturbed for a certain length of time, lividity becomes well developed and *fixed*, and does not alter in position even with subsequent alteration in the position of the body. This was till recently thought to be due to clotting of blood in the blood vessels. In fact, the word 'fixation' itself is erroneous. If the position of the body is altered after 'fixation' and left in the new position for some longer time, the intensity of the lividity gradually fades, even though some colour, probably due to haemolysis staining the tissues, may sometimes remain in the primary position. Blood is always found fluid in capillaries and venules, and this blood is responsible for postmortem lividity. Thus, clotting of blood is not responsible for the colour. 'Fixation' is an apparent phenomenon. If pressure is applied over the area of lividity, it blanches. After 6 to 8 hours, the lividity does not blanch, when it is said to be 'fixed'.

Colour of the lividity in certain cases helps in arriving at the cause of death. For example, it is cherry red in carbon monoxide poisoning, bright red in cyanide poisoning, blackish in opiate poisoning, etc.

Patches of lividity may sometimes be seen over non-dependent areas. The possible mechanism for this is the squeezing of column of blood in the deeper veins against gravity, towards the skin surface, by the muscles developing rigor mortis.[10] Such patches could be mistaken for contusion marks. This confusion is easily resolved by making an incision over the discoloured area. If it is contusion, staining of the tissue is seen which can not be washed off, while if it is lividity, the blood oozes out from the freshly cut blood vessels, and the fresh stain can be easily washed off with water. If still doubt persists, microscopic examination of the tissue from the suspected area clinches the diagnosis.

As the process of formation of postmortem lividity is purely a physical phenomenon, it is seen in the dependent parts of the internal organs also. It is important to bear this in mind, lest the appearance

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of it in the organs may be mistaken for some significant condition of medicolegal bearing.

Note:

- PM lividity disperses with the onset of putrefaction.
- PM lividity may not develop at all if the body is tossed and turned continuously as seen in fast flowing waters of rivers or the sea.
- PM lividity can not be appreciated well in dark coloured subjects.
- PM lividity may not be appreciable even in fair skinned bodies if they have bled profusely or were severely anaemic.

Significance of lividity in postmortem clocking is not very reliable. However, scientists have tried instruments utilizing light, to measure PM lividity and its application to establish the time since death, with some significant correlation between them.[11]

Evaluation of PM lividity using colourimeter-measuring system has also been tried with some success in postmortem clocking.[12]

Rigor mortis

During life, muscles are supple, and in a slightly contracted state. After passing through a phase of *primary flaccidity* soon after death during which they are totally relaxed, the muscles over a period of time, gradually stiffen. This stiffening is known as rigor mortis. Rigor mortis affects all the muscles in the body, both skeletal and smooth.

Rigor mortis is a physico-chemical phenomenon. Recalling one's memory of the elementary structure of the muscle is necessary to understand the mechanism of its formation. A muscle mass is made up of a number of bundles of long fibres. Each muscle fibre in the bundle consists of densely packed myofibrils, which are the contractile elements. These myofibrils are made up of two protein filaments, actin and myosin. Separation of actin and myosin are responsible for extensibility and softness of the muscles. Coming together of these actin and myosin filaments, which result in contraction is dependent on the energy provided by adenosine triphosphate (ATP).

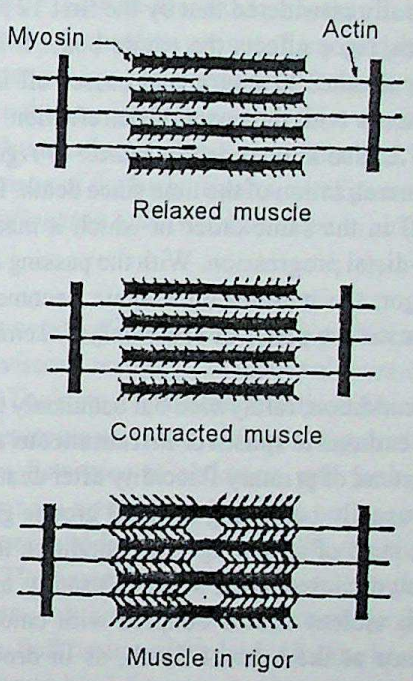

Fig 6.4 Schematic arrangement of myosin and actin filaments in the muscle fibre.
Adapted from **Gradwohl's** *Legal Medicine*. **1976.**

Constant supply of ATP is maintained in life. Failure of resynthesis of ATP after death results in the muscles becoming hard and rigid. However, some amount of ATP is still available after death till the glycogen reserve lasts. When once this glycogen reserve is depleted, there is no more ATP available, and the contractile elements are converted into dehydrated, stiff, gel-like mass resulting in the muscles themselves becoming hard and rigid.

Rigor mortis becomes noticeable first in the eye lids, then the muscles of the face, jaw and neck. Then it is seen to affect the upper limbs, thorax,

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abdomen and the lower limbs. Though rigor mortis is basically due to chemical changes, the physical aspect is responsible for this apparent proximo-distal progression, *viz.*, smaller muscles having less glycogen reserves than the larger muscles are first to be affected.

It is generally considered that by the first 12 hours after death, rigor affects the whole body. It is retained for another 12 hours, and passes off in the next 12 hours with the onset of putrefaction. This *Rule of 12*, also known as the *March of rigor*, is only a generalization of the time since death. Rigor passes off in the same order in which it made its proximo-distal progression. With the passing away of the rigor, the muscles once again become softened. This is the phase of *secondary flaccidity*.

Another condition, rarely seen but commonly talked about, is **cadaveric spasm** or **instantaneous rigor**, where instead of primary flaccidity after death, the muscles usually belonging to small groups go into a sudden state of stiffening. This condition though of unknown cause, when seen, is usually associated with violent deaths coupled with emotional disturbance at the time of death, as in drowning (where grass or weeds may be clutched in the hands), suicide by shooting (where the weapon may be tightly grasped in the hand), etc.

Other conditions that result in the body becoming stiff after death include thermal deaths (*pugilistic attitude*, page no. 147), and gases evolved during putrefaction. The stiffening of the body in deaths due to burns (*heat stiffening*) is due to coagulation of muscle proteins, while in exposure to severe cold (*cold stiffening*), the body becomes stiff due to freezing of the subcutaneous fat and the water content in the body, forming icicles. Gases evolved during the putrefactive process also stiffen the body (*gas stiffening*), but poses no problem in distinguishing it from rigor mortis.

Note:

- Application of force results in breaking of rigor, which does not set in again when once broken.
- As rigor is a chemical phenomenon, any condition that depletes ATP fast, will hasten its early development. For example, violent muscular contractions at the time of death due to epilepsy, strychnine poisoning, tetanus, electrocution, etc.
- In robust, healthy young individuals, it appears late because of good glycogen reserve in the muscle mass.
- As rigor mortis is not functionally related to the nervous system, it can develop in paralyzed limbs also.
- As rigor is also not dependent on blood supply, it develops even in amputated limbs.
- If a body is frozen soon after death, and such a body is brought back to normal environmental temperature, rigor mortis which had been arrested till then, will appear subsequently. Estimation of time since death from such thawed bodies becomes erroneous.
- Apart from postmortem clocking, rigor mortis helps in identifying any alteration in the position of the body, if the position in which the body has undergone rigor is incompatible with the position in which it was discovered at the scene.

LATE CHANGES

Putrefaction

Onset of putrefaction initiates the breakdown of complex organic constituents of the body into simpler inorganic substances. Such breakdown of complex structure is facilitated by *autolysis* and *bacterial action*, while the destruction of the body tissues by various fauna expedites the process of disintegration.

Autolysis: The digestive action of various enzymes released by the cells after death leads to softening and liquefaction of tissues. Soft tissues like the

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brain, lungs, pancreas, gall bladder, and stomach are affected earliest. Architectural patterns of the organs are lost. Maceration of the dead foetus inside the uterus is a classical example of autolytic putrefaction. This means the process of autolysis is independent of any bacterial action. It is an aseptic chemical process.

Bacterial action: Any condition conducive for rapid bacterial growth results in early onset of putrefaction. Bacteria disseminate and multiply fast after death, as stagnant blood offers an excellent medium for their growth. Apart from the normally inhabiting bacteria of the body, bacteria from outside also may enter the body through open wounds. These bacteria generally comprise streptococci, staphylococci, escherichia coli, proteus species, and clostridium welchii.

While autolysis produces limited gross change, bacterial action is the major component of putrefaction

Changes occurring due to putrefaction:

Greenish discolouration in the right iliac fossa is the earliest external manifestation of the onset of putrefaction, which occurs around 12 to 24 hours after death. This is the area where the caecum lies, the contents of which are more fluid and rich in bacteria. Hydrogen sulphide produced by the bacteria acts on the haemoglobin (that has now become extracellular due to haemolysis), to produce sulphhaemoglobin. This imparts the greenish hue. With the passage of time this greenish discolouration, which is very obvious in fair skinned bodies, subsequently spreads to the abdomen, and also makes its appearance in the chest, face, and the limbs. Coalescence of these coloured areas results in the entire body becoming greenish or greenish-black. Later, in about 36 to 48 hours, a network of veins becomes visible in the form of a characteristic mosaic like pattern (*marbling*), over various parts of the body like the root of the neck, shoulder, etc. This results from staining of the vessel walls due to diffusion of haemoglobin.

By the time the colour changes become visible externally, evolution of gases would have started internally. These gases possess very unpleasant smell. They include hydrogen sulphide, ammonia, carbon dioxide, carbon monoxide, methane, etc. These gases by themselves are not of any medicolegal significance, but the pressure effects they produce when they accumulate in sufficient quantity are so much as to bring about gross changes in the appearance of the body. Evolution of gases results in a) *ballooning effects* and b) *dispersion of p.m lividity*.

Ballooning effects: Gases easily collect in the hollow viscera resulting in their voluminous expansion. The stomach and the intestines which balloon out, increase the intra abdominal pressure resulting in distension of abdomen, and pushing of the diaphragm upwards. Lungs are squeezed by this pressure, and froth oozes from the mouth and nostrils. The gases not only accumulate in the hollow viscera, but also penetrate into every tissue and organ. Soft tissues of the body like the breast, scrotum, periorbital tissues, etc., increase in their size enormously. Eyeballs protrude out of the sockets, the cheeks puff out, and so also the lips. The tongue protrudes through the mouth, and on exposure to air becomes dark in colour. The facial features alter so much that even the resemblance to a normal human face is lost. At this stage, identification by the external features becomes extremely difficult even to the closest relatives.

Percolation of putrefactive gases into the undersurface of the skin results in the epidermal layer ballooning out, which resembles blisters caused by scalding. In contrast to protein rich fluid present in the blisters caused by scalding, putrefactive blisters contain only air. The epidermal layer from the hands and feet slip off like glove

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and stocking. It is important to retain and preserve this part of the skin as it can yield a full set of fingerprints that may help in establishing identity.

Increased pressure inside the abdomen may result in the expulsion of foetus in the case of a pregnant deceased. Uterus and rectum may protrude out due to pressure.

Though the evolution of gases may commence as early as 18 hours after death, a full-blown picture to develop may take 48 to 72 hours.

Dispersion of postmortem lividity: Evolution of gases moves the blood in the blood vessels. If the blood is clotted it now liquefies, and haemolysis takes place. Movement of this blood results in dispersion of p.m lividity in any direction. Therefore assessment of the position of the body at the time of death, based on lividity at this stage becomes difficult.

Entomology of the cadaver:

Emanating foul smelling gases of putrefaction attract various insects. There are eight successive waves of invasion from the beginning of putrefaction right upto skeletonisation.[13] Initially, two-winged flies predominate, but later there will be involvement of several other kinds of insects including beetles.Two-winged flies such as blue bottles, flesh flies, house flies, etc., lay their eggs in about 18 to 36 hours (sometimes as early as 6 to 10 hours).[14] Mostly these eggs are laid in the mucocutaneous junctions such as lips, nostrils, anus, vagina, or even in open wounds. The eggs hatch into larvae (*maggots*) within 12-24 hours. These larvae are very voracious eaters and they grow in size upto 4 to 5 days, during which time they devour the tissues and hasten the process of disintegration of the body. The maggots then enclose themselves inside shell-like structures (*pupae*), which in another 4 to 5 days break open to release young flies (*nymphs*). The study of the life cycle of these insects, which infest dead bodies, is known as **Forensic Entomology**. The role of the doctor while doing autopsies on such maggot-infested bodies is only to pick up these maggots, preserve them properly, and send them to a qualified entomologist for species identification. Maggots immersed in boiling absolute alcohol die instantly in extended position, which facilitates measurement of their length. Calculation of time since death is done as follows[15]:

Time since death = A + B X (cd)

where:

A is the stage of invasion (in hours, days, weeks, or months)

B is the stage of life cycle (in hours, or days) and

cd represents climatic factor correction.

Box 6.1 outlines some Indian cases where-in entomological study was successfully accomplished to arrive at the time of death.

Colliquative putrefaction: In 3 to 5 days after death, the nails and the hair become loose and can be pulled off with ease. The teeth become loose and can be pulled out from their sockets. The abdomen and the thorax may burst open. The sutures on the skull in children loosen, the liquefied brain matter oozing out through these deficiencies. With further progression of putrefaction, from 5 to 10 days, *colliquative putrefaction* ensues, resulting in the tissues and organs being converted into a soft, thick, black, pultaceous mass, totally losing their architecture. However even at this stage, organs such as the prostate and non-gravid uterus can be identified, as they resist putrefaction, thus helping in the identification of the sex.

This liquefied unrecognizable pultaceous mass gradually falls off from the bony attachments leaving the skeleton exposed. In 1 to 3 months, complete skeletonization occurs.

Sometimes the environmental conditions may arrest the progression of putrefaction and may devi-

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ate it to either *adipocere* formation or *mummification*.

Adipocere (Saponification; Grave wax): This arrests the further progression of putrefaction. Adipocerous change is due to hydrogenation and hydrolysis of body fat. The pre-requisites for adipocerous change of the body fats include a) abundant body fat, b) humid climate, c) still air, d) warm temperature, and e) bacteria producing fat-splitting enzymes, like Clostridium welchii. Thus a body lying on damp soil, in a shady place, with optimum environmental temperature, and no free air current blowing over it, undergoes adipocerous change. Adipocere has the appearance of rancid butter, and gives off a sweetish but disagreeable smell. Transformation of the body fat into adipocere is usually seen over areas such as cheeks, breasts, buttocks, or limbs, though rarely the whole body is affected. It takes generally about 3 weeks for adipocere to develop fully.

As the progression of putrefaction is arrested, the facial features are recognizable, which helps in identification of the deceased by friends or relatives. Similarly, as the wounds are also preserved without much alteration, the weapons used (and to a certain extent the cause of death), can be identified. The approximate time since death can also be estimated.

Mummification: In hot and dry climate, where there are warm air currents freely flowing over the body, putrefactive process becomes arrested and the body becomes mummified due to dehydration and desiccation of the tissues and the organs. The skin is dark and tightly adherent to the skeletal frame. Mummification usually requires 3 to 6 months. A mummified body does not emanate any foul smell.

Mummification helps in identification, as the general facial features are preserved. It also helps in recognizing the presence of wounds, and the estimation of the approximate time of death and place of death.

Note:

- Rate of putrefaction varies depending on whether the body is in an open place, buried, or in water. It is generally presumed that if the rate of putrefaction is considered as 1 unit of time on open ground, it is 2 units in water, and 8 units in deeply buried bodies. This is known as *Casper's dictum*.
- Evolution of gases in solid organs like liver leads to an alteration in the architecture visible to the naked eye. In such cases, the liver on cut section has a honeycomb appearance (*foamy liver*).
- Study of insect's life cycle is the job of an entomologist. The doctor should not guess the postmortem interval from his very limited knowledge of entomology.
- Usefulness of chemical analysis of maggots for poisons, which are not destroyed by the putrefying process, should be kept in mind.

ESTIMATION OF TIME SINCE DEATH

The time since death or *postmortem interval* is the period that has elapsed between death and the actual performance of the postmortem examination. The significance of such estimation of the time has been mentioned at the beginning of the chapter. Though the exact time of death can rarely be estimated on the basis of autopsy findings, an approximate range can usually be deduced by intelligent interpretation of the various changes that take place after death. Estimation of the time since death within a narrow range is possible only in the early period. Once putrefaction sets in, this range becomes wider (**Fig. 6.5**). However, entomological study can help in narrowing the range considerably.

Evidence for estimating the time since death generally falls under the following three categories[16]:

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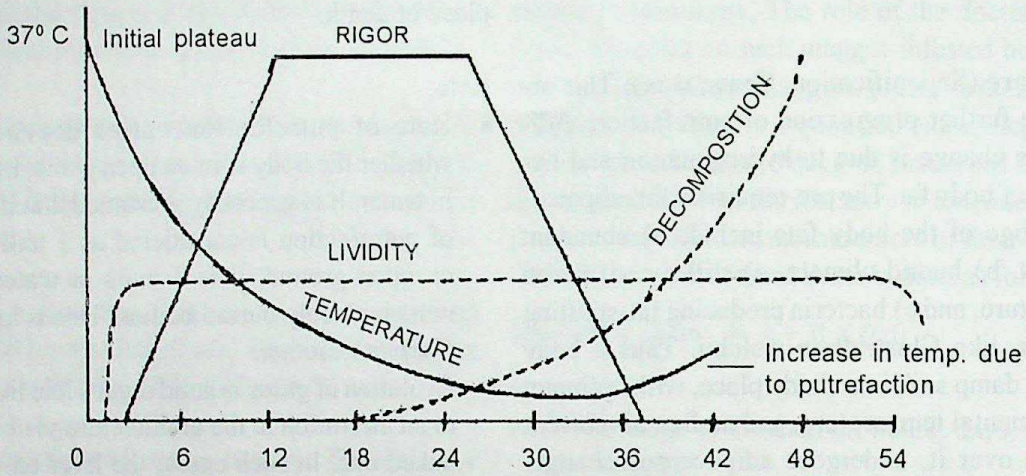

Fig 6.5 Graph showing the 'time since death' based on postmortem cooling, lividity, rigor mortis, and putrefaction.

- Corporal evidence – that which is present in the dead body.
- Environmental and associated evidence – that which is present in the vicinity and general surroundings of the deceased.
- Anamnastic evidence – that which is based on the deceased's ordinary habits, movements and day to day activity.

Assessing the time of death by environmental evidence and anamnastic evidence is mostly the responsibility of the investigating officer in India. The doctor is generally not involved. The doctor's assessment of time since death is almost exclusively dependent on the evidence that could be deduced by the findings on careful postmortem examination. Naturally he relies upon the changes that take place after death such as postmortem cooling, postmortem lividity, rigor mortis, and putrefactive changes, bearing in mind all those factors that alter the outcome of these individual changes. **Box 6.2** summarises the changes useful in computing the time since death.

Additional Methods - *Gastric emptying time* : If the time of the last meal is known, presence or absence of food and the state of digestion in the stomach helps to a certain extent in estimating the time since death. But the rate of emptying of the stomach depends on many variables including the type of food eaten, the emotional state of the person, any surgical procedure done on the stomach such as gastrojejunostomy, etc. Therefore this method must be used only as corroborative evidence.[17]

Bladder content : It is usual to empty the bladder before going to bed. On the morning of the death, if the body is found to be warm and supple and the bladder is full, it means that the person has died in the early hours of the morning, and hence the time since death is very short, probably with in the last 1 or 2 hours. On the other hand, if the body has developed a certain amount of rigor and is cold to touch, and the bladder is full, it means that the person must have died the previous night, probably before going to bed. This is fairly good corroborative evidence.

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Box 6.1 Forensic Entomology: Some Indian Case Studies

Case No. 1

Two elderly widows (both sisters) aged 80 years and 70 years, were found dead in their residence on Kingsway Road, Secunderabad, (Andhra Pradesh) on the morning of 8 March 1984, by their brother who immediately notified the police. Both women had been living alone for several years. Since stab injuries were noticed on the bodies, a case of double murder was registered. Both bodies were in an advanced stage of decomposition, and numerous maggots were crawling over them. The bodies were subjected to autopsy during the course of which some maggots were collected, killed instantly in boiling alcohol, and sent for entomological study. The larvae were subsequently identified as belonging to flesh flies (*Sarcophaga species*). Based on their development, and taking into account prevailing climatic factors, the time since death was computed to be 6 to 8 days. This information helped to convict the arrested suspect who had committed the murders in the course of robbery.

Case No. 2

An unidentified male aged about 70 years, was found dead inside a stationary railway carriage in the workshop yard at Secunderabad Railway Station, on 4 August, 1985, at about 10.30 a.m. by the Railway Protection Force. A case of unnatural death was registered initially by the Railway Police, and the body was subjected to autopsy which however disclosed natural causes (heart disease). In order to fix the time of death, a few maggots which were found crawling around the natural orifices were collected and submitted for entomological study. This revealed the insect species to be blue bottle flies (*Calliphora species*). Based on the stage of development and prevailing climatic factors, the time since death was computed to be 24 hours to 60 hours.

Case No. 3

A male aged about 35 years was found dead among some shrubbery, in the campus of the Central University of Hyderabad, at Ramachandrapuram, on the evening of 25 July 1985. The police was notified who registered a case of murder, since there were stab wounds on the body. During autopsy, maggots crawling around the injuries and natural orifices, were collected and submitted for entomological study. The flies were identified as belonging to *Sarcophaga species*, and the time since death was calculated on the basis of the stage of development and environmental factors, to be 4 to 6 days. This helped to convict the suspect who had murdered for revenge, since the deceased had been having an illicit affair with the former's wife.

Case No. 4

The deceased, a middle aged male who was suffering from tuberculosis, proceeded to Gandhi Hospital, Secunderabad, from his residence on the morning of 28 August 1985, since he had developed abdominal pain. He was accompanied by his daughter. At about 12 noon, both of them reached the hospital, where-upon the young girl was despatched by her father to fetch some articles from the nearby market, while he himself went towards the out-patient department. However on returning from the market, the girl could not locate her father, and went home alone. Later that night at about 8.00 p.m, a Municipal Corporation worker noticed the body of the deceased lying on an isolated stretch of footpath, at the rear of the hospital, and notified the police. A case was registered, and the

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body was subjected to autopsy the next day. While the cause of death could not be determined with certainty, it was presumed to be related to a worsening of his underlying condition (tuberculosis). Since some fly eggs were seen around natural orifices and in the scalp hair, entomological study was carried out to fix the time of death. The eggs were identified as those belonging to *Calliphora species*, (*Calliphora erythrocephala*). This fly generally does not lay eggs during the night. The size of the eggs coupled with the fact that larvae had not emerged, indicated that the eggs could have been laid 6 to 8 hours prior to the autopsy (held at about 2.00 p.m). Hence the eggs may have been laid between 6.00 a.m. and 8.00 a.m. on the morning of the autopsy.

In this particular case, all that could be deduced by the application of entomological knowledge was that the eggs could not have been laid the previous day as larvae would have been observed at the time of the autopsy. Hence death may have occurred sometime between 6.00 p.m and 8.00 p.m the previous night.

***Case No.1** is taken from the case files of **Dr. V K Kashyap**, presently Deputy Director of Central Forensic Science Laboratory, Calcutta. The **remaining cases** were part of doctoral (MD) research carried out by **Dr. V V Pillay**.*

Box 6.2 Postmortem Changes and Postmortem Interval

Condition of the body	Time since death
▪ Warm supple body, transparent cornea, no patches of postmortem lividity	*within one hour*
▪ Patches of lividity on the dependent parts, rigor mortis appreciable in the muscles of the face.	*2 to 3 hours*
▪ P.m. lividity well developed and 'fixed', rigor mortis at least in the upper part of the body, body surface cold	*6 to 8 hours*
▪ Rigor mortis all over the body, with or without greenish dicolouration over the right iliac fossa, body cold	*around 12 hours*
▪ Body cold and stiff, greenish discolouration over the right iliac fossa, eggs of flies	*12 to 24 hours*
▪ Body is cold, rigor has receded, greenish discolouration over the abdomen, and chest, distension of the abdomen with evolution of gases	*24 to 36 hours*
▪ Marbling, distended abdomen and thorax, presence of maggots, post mortem blisters	*36 to 48 hours*
▪ Whole body bloated, face unrecognisable, nails and hairs easily pulled off, grown maggots or pupae all over the body	*3 to 5 days*
▪ Colliquative putrefaction where the internal organs are reduced to black unrecognisable pultaceous mass	*1 week*
▪ Most of the soft tissues gone, organs that putrefy late like prostate and nongravid uterus recognizable	*2 weeks*
▪ Skeleton exposed bare	*1 to 3 months*

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Biochemical changes: Though a lot of research work has been undertaken to estimate the time since death based on biochemical changes such as changes in the potassium content of the vitreous humor, level of lactic acid and non protein nitrogen of c.s.f, and a plethora of other chemical parameters, they are mostly only of academic interest. As the antemortem values of these chemicals vary substantially, they are of limited practical value.[18]

Circumstantial evidence: If the body is found in an open place, the state of grass underneath the body helps in the estimation of time since death. As the grass becomes deprived of sun light for photosynthesis they gradually turn yellow, due to conversion of chlorophyll to xanthophyll. The usual time required for such a change in colour is 3 to 5 days.

Uncollected mail or news papers, recent purchases of household materials with dated bills, condition of the food in the kitchen, etc., are other factors which can be taken into consideration in assessing the time since death.

Numerous other methods have been propounded to estimate the postmortem interval,[19] which are however beyond the scope of this book.

In all cases of unwitnessed unnatural death, if the estimation of the time since death is critical in checking out an alibi, the doctor should base his opinion only after correlation of evidence from all the sources available, along with a critical analysis of the validity and reliability of these findings.

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CHAPTER - 7

Medico-Legal Autopsy

"A surgical operation is attended with pain, and is for the benefit of the individual, An autopsy is free from pain, and is for the benefit of humanity".

- Brouardel

Though **necropsy** is the most accurate term for the investigative dissection of a dead body, the term **autopsy** is used more commonly. **Postmortem examination** is an alternative expression, but it unfortunately suffers from a lack of precision about the extent of examination, for in some countries many bodies are disposed of after external examination without dissection.[1] The first autopsy is said to have been performed in the year 1559, when King Henry II suffered a fatal injury and died ; eleven days later **Dr. Ambroise Pare**, a celebrated French surgeon dissected the body and discovered a subdural haematoma.

A **medicolegal autopsy** is performed only on the instructions of a legal authority responsible for the investigation of sudden, suspicious, obscure, unnatural or criminal deaths. In India, such legal authority may be represented by a police officer or a magistrate. The information so obtained is invaluable in the administration of justice, in convicting criminals, and in protecting innocent suspects. On the other hand, a **clinical** or **pathological autopsy** is performed in death due to natural causes, in order to ascertain the exact cause, if the diagnosis made by the treating physician is to be confirmed or refuted, or if diagnosis could not be arrived at all while the patient had been alive. The consent of the next of kin is essential, while there is no need for a requisition from the police or the magistrate.

A special type of autopsy is the **psychological autopsy** which is not really an autopsy at all, but instead refers to the perusal of medical and personal history, and other background information of a deceased person who had committed suicide, in order to arrive at the motive for the act.[2]

It has been suggested that if a conventional autopsy is difficult or impossible to undertake for any reason, a postmortem endoscopic examination can be performed with the help of a trocar and telescopic device coupled to a video camera.[3] This is referred to as **endoscopic autopsy**. But there are serious limitations to this technique, and hence is not generally recommended.

OBJECTIVES OF A MEDICOLEGAL AUTOPSY

1. To establish the identity of the deceased.
2. To ascertain the cause and manner of death.
3. To determine the nature of the weapon or causative agent.
4. To determine the time of death.

RULES

- Before beginning the autopsy, the body must be properly identified by the police officer who accompanies the body. In the case of an unidentified body, photographs and finger prints should be taken by the police.

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- The medical officer should ascertain the background of the case before starting an autopsy. Inquest reports and hospital records are helpful in this context.
- The autopsy must be done in a well equipped mortuary, by a qualified person with requisite knowledge and experience. However, occasionally it may become necessary to do an autopsy at the site where it was discovered, when the body is in an advanced state of putrefaction. This is because materials of evidential value may be lost in transit, if the body is transported. Delay in performing an autopsy must be avoided as far as possible.
- The medical officer should try to limit the number of those present in the autopsy hall to a minimum. Not only is there a greater risk of loss of confidentiality, but sheer numbers can make the mortuary over crowded. This hampers movement, causes distraction, and adds to the risk of infection and contamination. No one should be present merely as a casual observer, not even senior police officers (if they are not directly involved in the investigation). With the increased sophistication and complexity of forensic and police procedures, more and more people cram into a mortuary cluttered with cameras, video recorders, scene-of-crime kits, etc., that there is sometimes no room for the medical officer himself to move!
- When the deceased has been under medical care before death, it is acceptable practice to allow and encourage the physician concerned to be present at the autopsy, as he is in possession of relevant (and sometimes vital) medical history.
- All persons present at an autopsy should be listed and named by the medical officer in a separate register, or on the autopsy report.
- Autopsies are best done in day light, otherwise colour changes relating to bruises, jaundice, etc., are likely to be missed.
- Detailed notes should be made at the time of autopsy. It is better to dictate the notes to an assistant while the autopsy is in progress. Subsequently, the medical officer must verify the notes and sign it. All corrections should be initialed. Nothing should be erased. If no assistant is available, the doctor should prepare the report immediately after completion of the autopsy. A tape recorder may be used. Sketches should be made of all major injuries and lesions.
- Every medico-legal autopsy must be thorough, meticulous, and complete. A partial autopsy should never be done. Useful information can be obtained through careful observation, even in grossly decomposed bodies.[4]

PROCEDURE

External examination

This must be thorough, and refers to general description regarding build, height, weight, age, sex etc., of the deceased, as well as details of deformities, personal belongings, etc., in unidentified bodies.

Clothing should be examined for stains, cuts, tears, and trace evidence. Natural orifices such as mouth, nostrils, ears, rectum, and vagina should be examined, and swabs collected if necessary, for bacteriological or chemical examination.[5]

Postmortem changes such as the state of rigor mortis, postmortem lividity, body temperature, and putrefactive changes, should be assessed and noted, which will help in ascertaining the time of death, and whether the body had been tampered with after death. Wounds (if any) on the body are examined and described, regarding their nature, situation, size, shape and direction. It must be mentioned as to whether the wounds are antemortem or postmortem in origin.

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Signs of disease indicated by pupillary changes, colour changes, bleeding in the eyes, skin eruptions, emphysema, or oedema, should be noted.

There is no substitute for a good colour photograph to preserve the appearance of a wound or injury. If blood spots or smears are present on the skin, the area should be photographed before and after the skin is cleaned. Excepting stab and firearm wounds, all other injuries should be divided into *external injuries* and *internal injuries*. Specific body parts should be examined for deformities, i.e., fractures or dislocations, and confirmed by dissection.

A list should be made of all articles removed from the body, and mentioned in the report, before handing them over to the police in a sealed cover.

The postmortem report should include details of all surgical procedures and therapeutic measures evident on external examination, or from the case sheet, in "treated" cases. All external injuries found on the body should be compared with those noted in the inquest report, and any discrepancy noticed should be highlighted.

Internal examination

The three major body cavities must always be examined in detail. However, the spinal column need not be opened if there is no specific indication. In case of suspected poisoning, certain viscera and body fluids must be preserved for chemical analysis, (*see page no. 295*).

In general, it is better to begin by examining that part or cavity suspected to be affected most.

However, before dissecting the neck, it is ideal to drain away the blood by opening the cranium and other body cavities, in order to avoid artefacts.

Incisions: (Figs 7.1 & 7.2)

For the trunk

1. *I-shaped incision* - The usual incision is a straight line from the chin to the pubis (symphysis menti to symphysis pubis), with a deviation at the umbilicus in order to avoid cutting through it. The skin over the neck and trunk can thus be reflected laterally on either side.

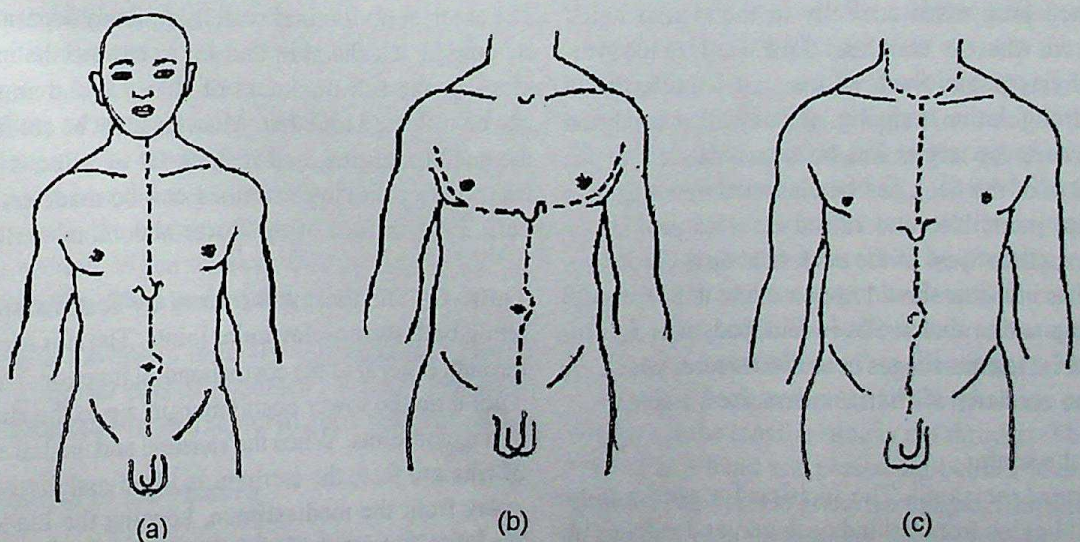

Fig 7.1 Trunk incisions

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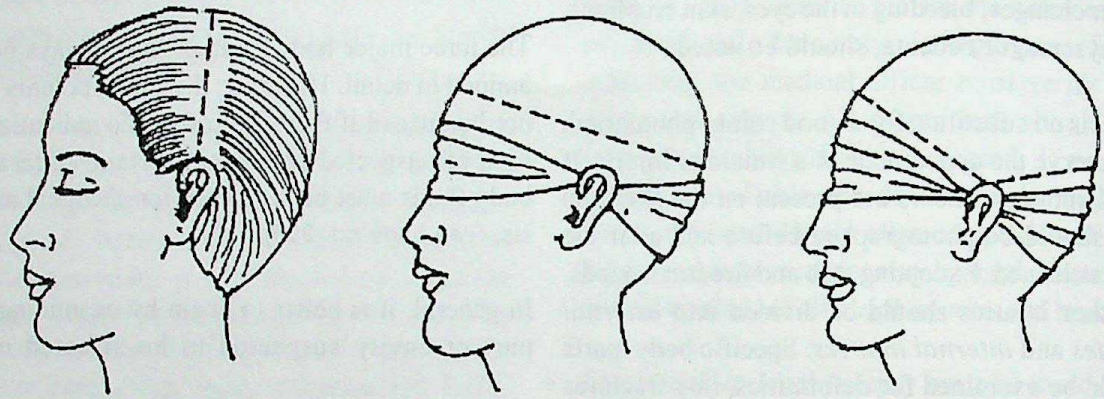

Fig 7.2 Scalp and skull incisions

2. *'Y' shaped incision* - begins at a point close to the acromial process and extends down below the breast, and then medially across to the xiphoid process. A similar incision is made on the opposite side of the body. From the xiphoid process, the joint incision is carried downwards in a straight line to the pubis.

3. *Modified Y-shaped incision* - This incision begins behind each ear from the mastoid, and extends down to the mid-point of the clavicle, and then progresses medially to the sternal notch from where it continues downwards to the symphysis pubis. Such an incision is preferred in strangulation, hanging, and any other condition where the larynx has been traumatised, as the skin of the neck can be dissected upwards upto the mandible, and raised clear to provide an excellent view of the neck structures. However this incision should not be made until the skull cap and brain have been removed, to avoid artificial haemorrhages in neck structures which can be confused with true antemortem trauma.[6]

For the scalp

Coronal incision The incision for access to the skull begins from behind each ear, and extends upwards on either side to meet over the crown of the head.

Opening the body cavities

The skin, subcutaneous tissue, and fat are flayed laterally from the main incision, taking care not to extend beyond the lateral edge of the neck, or the outer third of the clavicles. Over the thorax, the tissues (including the pectoral muscles), are flayed to the mid axillary line in the upper part, and in the lower part, even further posteriorly towards the costal margins.

The anterior abdominal wall is similarly separated, stripping back the skin and fat to expose the muscles - or the full thickness of skin, fat and muscle can be reflected together. Muscles must be cut from the costal margins, and if thick fat overlies, a few transverse relieving incisions can be made on the peritoneal surface of the lower abdominal wall.

Thorax - The thorax is opened by first disarticulating both sterno-clavicular joints. The ribs are cut through (lateral to the costochondral junctions) from a point on the lower costal margin, up to the sterno clavicular joints. When the sternum and medial ends of ribs are free, the sternum is lifted and dissected away from the mediastinum, keeping the blade of the knife close to the bone, in order to avoid cutting the pericardium. The sternum is examined for frac-

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tures or other lesions, before it is put aside. The whole of the thorax and abdomen is now open for inspection. The degree of inflation of the lungs should be assessed, noting complete or partial collapse, emphysema, and over-distension.

If pneumothorax has been suspected before hand, a postmortem radiograph is the best confirmation. Alternatively, the chest wall can be punctured in the mid-axillary line, after filling the pocket between the ribcage and reflected skin with water. If bubbles escape into the water, it is indicative of pneumothorax.[7] The test cannot succeed if there is a patent communication between pleural cavity and the bronchial tree.

The pleural cavities are examined for adhesions, effusions, pus, blood, fibrin, and even for extravasated gastric contents.

Abdomen - The abdomen is inspected for evidence of ascites, faeculent fluid, pus, or blood.

The omentum may show inflammation or fat necrosis, or it may be totally devoid of fat (as in starvation deaths). The coils and loops of bowel are examined for any abnormalities, e.g., infarction, peritonitis, distension, etc. *Beware of mistaking postmortem hypostasis for necrosis of mesenteric embolism or strangulated bowel.* Though the dark colour may be similar, hypostasis has irregular segments when the gut is stretched out, where-as in infarction the discolouration occupies one continuous section. When well established, the bowel wall will be lustreless and friable.

Finally, the bowel is gently moved aside to look at the posterior part of abdomen : a retro- peritoneal haemorrhage from a ruptured aorta may be present.

Removal of the viscera

After the body cavities have been inspected, the organs are removed enmasse (**Rokitansky method**).[8] First, the intestines are removed. The greater omentum is lifted upwards to expose the coils of small intestine. The upper most part of the jejunum is identified, where it passes retro-peritoneally to join the terminal duodenum. Here, the mesentery is perforated with a knife, and the gut is cut through between double ligatures. The mesentery is then stripped off the entire intestinal length until the ileo-caecal valve is reached.

The caecum is then mobilised medially using traction, with minimum use of the knife (to avoid puncturing it). When the hepatic flexure is reached, the omentum is pulled downwards to draw the transverse colon tense against the mesocolon, which is cut through taking care not to open the adjacent stomach. The splenic flexure is then pulled medially and downwards, and the descending and sigmoid colon are separated from the posterior abdominal wall. The upper rectum is cut between double ligatures.

Removal of neck structures

To make the removal of neck structures easier, a wooden block (10 to 15 cm high) should be placed under the shoulders of the cadaver which allows the head to fall back, and thus extends the neck. This must be done gently to avoid the well known *undertaker's fracture*, which is a subluxation of the lower cervical spine, due to tearing of intervertebral disc between C_6 and C_7. There is danger of misinterpreting this as an antemortem injury. The neck structures are now freed by passing a knife under the skin of the upper neck until it enters the floor of the mouth. The knife is then run around the inside of the mandible to free the tongue. The tissues at the back and sides of the pharynx are divided, and the tonsilar area is cut through. The fingers of one hand are passed up behind the mandibular symphysis to grasp the tongue, which is then drawn down, the remaining tissues behind the larynx being divided to release the neck structures.

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This should be done as far laterally as possible, so that the carotids can be removed along with the laryngeal structures. The pharynx and glottis should be examined at this stage to look for any obstruction, bleeding, or any other abnormality of the upper airway.

Removal of thoracic contents

The subclavian bundles of vessels and nerves are divided by passing the knife from the inside of the thorax (around the medial ends of the clavicles and first ribs), to release the trachea and oesophagus. With gentle traction, the neck structures are held up and pulled caudally, while carefully clearing all attachments to the thoracic spine with the knife.

Traction should be minimal and, as soon as the thorax is entered, the hand should move from the neck structures to place two fingers under the upper lobes of the lungs, lifting them and the mediastinum, as the knife clears the midline structures down to the diaphragm.

If the neck structures are pulled too hard to drag out the thoracic viscera, they may get ripped off. In addition, the descending aorta may suffer transverse intimal tears from traction, which can resemble the genuine "ladder tears" seen in many traffic accidents.

Pleural adhesions may prevent clean removal of the lungs. If these are minimal, they can be cut through. If the whole pleural cavity is obliterated by adhesions, the lungs may be pulled away by making a cleavage plane with the hand, thereby stripping off the adhesions.

Removal of abdominal contents

When the chest organs have been cleared, they are laid back in the thoracic cavity, and the diaphragm is incised. The left leaf of the diaphragm is stretched, while the knife cuts through it laterally, near the costal margin. The cut should curve posteriorly under the organs to reach the spine, where it must pass through the cruciate ligaments, and then progress caudally behind the kidney which is mobilised forwards.

The knife cut then curves up over the psoas muscle and ends at the brim of the pelvis. The same is done on the opposite side. The thoracic organs are now lifted and gently pulled free of the cavity along with the abdominal viscera. Any resistance is due to incompletely cut cruciate ligaments which must be severed. The iliac vessels and ureters are cut through, and the whole bulk of viscera is removed to the dissecting table, where running water and adequate illumination must be available.

Removal of pelvic contents

The bladder is cut open, and the mucosa and trigone inspected, before the prostate is incised for examination. The testis on each side is pushed upwards through the inguinal canal, which is widened with the knife.

In a female dead body, the ovaries are incised and the fallopian tubes are examined, before the uterus is sliced in the midline from fundus to cervix.

Removal of cranial contents

After incising the scalp across the posterior vertex, from the mastoid process of one side to the corresponding mastoid process on the other side, the tissues are reflected forwards to the lower forehead, and backwards to the occiput. If head injury is present or suspected, the scalp should be reflected right back to the nape of the neck, paying attention to the tissues behind and below each ear to avoid damaging the vertebral and basilar arteries.

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The skull is sawn through, using either hand-saw or power tools. The sawing should preferably be angled, with a horizontal cut from the forehead to a point behind the ears, joined by a second cut passing diagonally upwards over the occipito-parietal area. This will prevent the skull cap slipping out of position, after it has been replaced and the scalp has been sutured, at the conclusion of the autopsy.

The calvarium (skull cap) is removed by leverage after cutting through completely. Hammer and chisel should not be used in forensic autopsies, even to ensure that the dura remains intact. The risk of extending an existing fracture or even causing new fractures because of hammering, is too high merely to justify an unmarked dural membrane. A cut dura is easily recognised as such by a pathologist. What is more important is to inspect the surface of the exposed dura and brain for evidence of oedema, bleeding, or inflammatory conditions. The skull cap is carefully inspected for fractures, and the dura is peeled off from its inner apect to study the inner surface.

To remove the brain, the superior sagittal sinus is first incised with fine scissors or scalpel, and then examined for thrombosis. The dura is cut all along the line of cleavage of the skull and folded back. Look for subdural or subarachnoid haemorrhage. Subdural hemorrhage can be easily washed away, where-as subarachnoid haemorrhage cannot be washed off. Examine the leptomeniges for inflammatory changes. Free the falx-cerebri from the cribriform plate and fold it backwards. Lift the frontal lobe of the brain by inserting the fingers of one hand under it. The optic chiasma and the cranial nerves are now identified and cut one by one, as far away from the brain as possible. Cut the tentorium on either side, and pass a narrow pointed knife through the foramen magnum, to cut the cervical cord well below the medulla.

Remove the brain gently with both hands. Strip away the remaining dura from the base of the skull and inspect for fractures. The brain is weighed before dissection.

Removal of spinal cord

This is not normally done. It is undertaken only if there are specific indications. The posterior approach is usually preferred in an adult.[9] The body is placed in the prone position and a midline incision is made from the occipital protuberance to about the 4th lumbar vertebra. The skin and underlying tissues are dissected away on either side of the vertebral spines, and the laminae are exposed. These are divided as close as possible to the transverse processes by means of an adjustable double-blade saw. A transverse incision is made at the lower end of the midline exposure to enable the dissector to grasp the lumbar end of the freed spinous processes with the bone forceps. The spinous processes can be lifted upwards in one piece.

The spinal cord enveloped by the dura mater is now exposed and inspected. The dura is gripped with forceps and drawn aside gently to expose the extradural nerves, which are divided on either side. The dura is picked up with the forceps so as to raise the cord gently, allowing the dissector to cut the remaining connections. The spinal cord and dura mater are now divided as near the foramen magnum as possible and removed.

Dissection of viscera

Brain

Examine the brain surface for evidence of injury, infection, or bleeding. Identify and scrutinise the circle of Willis. The brain is now placed on a flat board with the convexity in view, and the frontal pole away from the dissector. The hemispheres are separated with the left hand. A brain knife is placed

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in the longitudinal sulcus, and horizontal sections are made on either side just above the level of corpus callosum. The white matter of the brain is examined with particular reference to petechial haemorrhages, often seen in death from fat embolism. The lateral ventricles are now opened by incising the white matter with a scalpel. Inspect the choroid plexus and locate the interventricular foramen. The fornices and corpus callosum are cut and bent backwards. Examine the thalamus and caudate nucleus. The third ventricle is now exposed. Pass a probe through the aqueduct of Sylvius. Expose the fourth ventricle by cutting along the vermin in the midline.

With a long knife make parallel horizontal incisions, a little below the incision already made on the cerebral hemisphere. The internal and external capsule, as well as the basal ganglia are now exposed and examined. The cerebellum and brainstem are removed. Each cerebellar hemisphere is incised to expose the dentate nucleus. Serial sections are made through the pons, medulla, and the remaining cord.

If the brain is oedematous, the gyri appear flattened and the sulci get narrowed. Tentorial herniation produces a groove (Kernohans notch) on the surface of mid brain. Foraminal herniation produces indentations of medulla and cerebellum.

Spinal cord

The spinal cord along with its coverings is stretched out on the dissecting board. The dura is opened by scissors and forceps in the midline, the cord being exposed and its membranes and outer surface inspected. The cord is sectioned by a series of transverse incisions about half an inch apart and the cut surfaces inspected. It is advisable to dip the knife in water before commencing this dissection, as the cord will otherwise stick to the blade.

Other viscera

The thoracic and abdominal viscera are laid on a cutting bench under good illumination. Wash the viscera with ample water to flush the tissues as the dissection proceeds. Some pathologists maintain that this should not be done, as water can affect the quality of histological sections, a view which is debatable. In any case, the gross appearance of organs is usually far more important than microscopic findings, in most forensic autopsies.

Structures of the mouth and neck

The tongue is examined for disease or injuries. It should be sliced to detect deep haemorrhages (seen in strangulation), mostly at the sides and centre of the mid-part of the tongue. Gross congestion, which may be due to either pressure on the neck or to other congestive modes of death, is usually evident in the posterior part of the tongue. The tonsils and pharyngeal walls are inspected.

The glottis is examined for mechanical or infective obstruction, and the hyoid and thyroid horns are palpated for fractures. The oesophagus is opened up to the cardiac end of the stomach, and any suspicious material present such as capsules, tablets, etc., are retained for analysis.

The carotid artery on each side is slit-opened, and if thrombosis is suspected, the intracranial part should be examined in the cavernous sinus. The thyroid gland should be sliced and inspected. The trachea and main bronchi should be examined for disease or obstruction. Gastric contents are often found as a postmortem artefact ; it should not be assumed that antemortem aspiration has occurred merely due to the presence of gastric contents in the air passages.

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Lungs

The lungs are removed, and carefully examined for evidence of patchy collapse, emphysema, or petechiae (especially around the hilum and in the interlobar fissures).

Both lungs are weighed. Then each is laid on the dissecting board with the hilum facing down. Each lung is grasped by the left hand and cut across in the sagittal plane from apex to base, with a large brain knife wielded parallel to the board. This produces an antero-posterior slice, the lower medial part carrying the hilum. The two halves can now be opened like a book, and the surfaces examined for oedema, tumour, pneumonic consolidation, infarction, trauma, abscess, etc. The smaller bronchi must be examined for mucosal thickening, infection, or blockage. The smaller pulmonary arteries may reveal thrombosis or embolism that was not visible in the larger vessels.

Heart

There are almost as many ways of examining the heart as there are pathologists, and each operator must therefore decide upon the method which appeals to him most. One of the commonly followed methods is described here.

A pair of scissors is used to cut open the common iliac artery and extended right up to the aortic arch, and further beyond upto a few cms above the aortic valve, staying outside the reflection of pericardium. The interior of the aorta is studied for evidence of atheroma, aneurysms, or trauma. The inferior vena cava is opened from its lower end upwards. The pericardium is examined through a small nick for the presence of fluid or blood, then opened widely with scissors. The heart is delivered through the incision and inspected externally for adhesions, discolouration, pallor, or aneurysmal dilatation. In a child, the thymus is examined and dissected out at this stage.

The heart is removed by holding it up with the left hand so that its attachment is tensed against the neighbouring structures. A long knife is passed horizontally across the reflection of the pericardium cutting through the roots of aorta and other great vessels.

The detached heart is washed and placed in the anatomical position on the dissecting board, with the apex facing the operator and the anterior surface upwards. It should not be weighed until all blood clot contained in the chambers has been removed. The general size and shape should be noted. Any dilatation or thickening of the pulmonary conus should be noted as an index of right ventricular hypertrophy, especially if striae of transverse muscle fibres are present.

The right atrium is then opened. The interior of the atrium, as well as the septum and tricuspid valve are examined.

The interventricular septum is identified externally, and a cut is made about 1.5 cm to the right of and parallel to the septum over the right ventricle, which should be deep enough to enter the chamber. The cut must run up through the pulmonary conus and into the pulmonary artery. It is also extended downwards to the apex of the ventricle. A pair of fine scissors is now used (guided by the fingers of the left hand) to pass and cut into the tricuspid valve from the opened atrium. The whole of the right side of the heart can now be opened and displayed. It should be washed out, and the endocardium examined for lesions. The patency of the valve is tested under running water.

A similar procedure is followed on the left side. The scissors is introduced into a pulmonary vein and passed horizontally across to an opposite vein, thus opening the left atrium. Two fingers are introduced into the mitral valve to estimate its size and detect any stenosis.

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The heart is restored to the anatomical position and a cut is made parallel to the septum on the left side, going deep as the left ventricle is thicker. Guided by the fingers still in the mitral valve, the cut is extended upwards through that valve and out from the top of the atrium.

Various estimates of the normal weight exist for the human heart, but there is considersble variation depending on the sex and body weight. As a rule of thumb however, we can accept upto 380 gms as normal in an adult man of average build.

After weighing the heart, the endocardium and valves are examined in more detail. The coronary arteries must be scrutinised with reference to narrowing of lumen or stenosis. The ostia are examined for congenital variations (which are frequent), and for obstruction. Serial cuts are then made at intervals of not more than 3 mm, first into the left coronary artery, followed by the circumflex branch laterally, until the vessel becomes too small as it dips down from the epicardium to become intra muscular. The anterior descending branch is then followed down the front of the septum almost to the apex.

The right coronary artery is dealt with in the same way, beginning from the point where the right ventricle was opened, transecting the artery in its mid part. Serial cuts are made back to the aorta, and then the distal segment is followed laterally until it becomes the posterior descending branch. During this process, the dominance of right versus left vessels is noted.

Difficulty arises when severe calcification exists in the coronary arteries. The only solution then is to decalcify the vessels. Postmortem angiography is the other alternative, but if the autopsy case load is high, this can be difficult to accomplish.

Abdominal and pelvic organs

The stomach is washed externally in a stream of water and a small cut is made in the greater curvature, which is then opened widely with scissors. The contents are collected into a container and the gastric mucosa is examined for signs of inflammation, erosion, ulceration, etc. The contents are examined with reference to the nature, quantity, state of digestion, presence of foreign matter, smell, etc.

The liver is dissected out, weighed, and scrutinised. Serial sections are made along the long axis, and the cut surfaces are examined for evidence of congestion, fatty change, cirrhosis, abscesses, etc.

The pancreas is not ordinarily examined in detail, unless there is a specific reason. In relevant cases, look for calculi in the duct system or acini, evidence of fat necrosis, etc.

The adrenals are examined next, the right one being located on top of the right kidney, while the left is situated on the medial side of the left kidney. The amount of cortical fat and presence of bleeding or other abnormality is noted in each gland.

The kidneys are exposed by incising their capsules. They can usually be peeled out of their capsules unless these are adherent. The renal vessels can be examined when the aorta is opened. The kidneys are detached at their hilum and weighed, then cut lengthwise to examine the interior. The width of the cortex is about 1 cm in a healthy subject. The granularity of the surface and the clarity of the cortico-medullary junction is assessed, as well as the size of the renal pelvis.

The spleen is removed by cutting through its pedicle, and is sliced after weighing.

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The small intestine can be examined now. However, it is not customary to open the entire length of the small bowel, unless there is some specific indication.

In a female dead body, it is important to examine the uterus after removing it along with the fallopian tubes, ovaries, and a portion of the vagina. The cervix is scrutinised for evidence of ulceration, erosion, laceration, mucosal plug, etc. The uterine cavity is examined for products of conception, blood clots, and foreign bodies. Both the ovaries must be incised to look for corpus luteum, congestion, cysts, etc.

In a male dead body, the prostate may have to be examined, though this is not usually done. Testes must be examined in sudden, unexpected deaths, especially if the victim had died in the course of a brawl or assault. The scrotum should not be incised; instead an internal approach can be made via the inner aspect of the inguinal canal.[10]

FOETAL AUTOPSY

Objectives

- Determination of live birth and viability in the case of dead new-born infants.
- In suspicious deaths, to find out the cause of death.

  Autopsy is undertaken only after receipt of requisition.

External examination

This should be thorough, beginning with an examination of the coverings (if any), enclosing the foetus. Description regarding postmortem changes such as rigor mortis, postmortem hypostasis, and putrefaction must be given as usual (*vide supra*). Mummification or maceration if present, is described in detail. Natural orifices such as mouth, nostrils, ears, rectum, and vagina are examined.

Loss of alignment and over-riding of the bones of the cranial vault may be present due to shrinkage of cerebrum after death of the foetus, in cases of intra-uterine death (**Spalding's sign**).

Look for injuries over the body, particularly in and around the mouth and nostrils, and over the neck.

Examine the shape of the chest, noting whether it is arched or flat. Look for *caput succedaneum* over the head. Note down the following measurements : weight of the foetus, crown to rump length, crown to heel length, circumference of the chest, and circumference of the abdomen.

Make an incision in the web of the foot between the 3rd and 4th toes, so as to bisect the foot longitudinally, and extend into the ankle joint. Look for the ossification centres of calcaneum, (5th month), talus (7th month), and cuboid (9th month).

Make a horizontal incision across the front of the knee joint. Reflect the skin flaps, cut the muscular attachments, and reflect them as far away from the joint as possible using a scalpel. Make horizontal slices of the lower end of femur. Look for the ossification centre which should appear at about the 9th month of intra uterine life.

Similarly, slice the upper end of tibia. Look for the ossification centre which appears at birth. When the sternum is removed, it is examined in turn as to the status of its ossification centres. Bisect longitudinally, and look for separate centres for the manubrium, and the four pieces of the body. Describe them in sequential order with reference to the number, size, etc. If the gestational age of the foetus is less than 5 months, no centre will be seen in the sternum.

Internal examination

All the three major cavities should be examined. The body is opened by the usual I-shaped incision.

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The skin, subcutaneous tissue, and fat are flayed laterally from the main incision.

The ribs are cut through at the costocondral junctions, from a point on the inferior costal margin up to the sterno-clavicular joints. When the sternum (along with the medial ends of the ribs) is free, it is lifted and dissected away from the mediastinum, keeping the blade of the knife close to the bone to avoid cutting the pericardium. The sternal plate is placed aside. The whole of the thorax and abdomen is now open for inspection. After the body cavities have been examined, the organs are removed enmasse. Each organ is weighed, examined, and the macroscopic findings are recorded.

Note the position of the diaphragm in relation to the level of the ribs, which should be about the level of 4th or 5th rib if respiration has not taken place, and at the level of 6th or 7th rib if the child had breathed. This can however be affected by gases of decomposition.

Examine the lungs. Breathing causes permanent changes in the lungs, the extent of which depends on the depth and period of respiration. Whether the child was born alive or not, the lungs will fill the thorax. However, fully respired lungs completely fill the pleural cavities, and the medial edges overlap the mediastinum and part of the pericardium. Before respiration, the margins appear sharp, which become rounded when breathing has been established. Glistening bullae appear along the margins when there has been a struggle to breathe due to mechanical obstruction. Before respiration, the lungs appear dense, firm, and non-crepitant (like the liver). After respiration, they become soft, spongy, elastic, and crepitant.

Each lung may be weighed and subjected to the **hydrostatic test**, where-in floatation is tested in a pan or bucket of water. This is followed by individual lobes of lungs. After this, each lobe is cut into 8 to 10 pieces, and each bit is tested for floatation. If they also float, rule out the influence of putrefactive gases by pressing the lung bits in between two smooth wooden blocks Try the floatation test again. A positive hydrostatic test in the absence of putrefaction, is a reliable sign of respiration having been established. If only a few pieces float, it indicates incomplete aeration of the lungs which could be due to feeble respiration, or artificial inflation of the lungs. Pathological conditions such as pneumonia, pulmonary oedema, and atelectasis also may give rise to mixed results. If all the lung bits sink, it indicates that respiration has not occurred.

Collect tissue bits in 10% formalin from different areas of lungs for histological study -presence of hyaline membrane indicates live birth. Also, a respired lung will demonstrate expanded alveoli lined by flattened epithelium, and patent sections of blood vessels.

Make a coronal incision on the head from behind one ear to the other, reflect the scalp, and examine for injuries, cephalhaematoma or caput succedaneum, and for the presence and closure status of fontanelles. Examine the skull bones for fractures, relfect the dura, remove the brain, and examine for evidence of injury or disease. Remove the dura from the base of the skull and examine for fractures of the base.

Record any additional observations if present, for instance, internal signs of maceration, putrefaction, etc.

In cases where the cause of death is not clear, preserve relevant viscera and body fluids for chemical analysis. Preserve a sample of blood for peripheral smear, grouping, etc.

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ANCILLARY INVESTIGATIONS IN MEDICO-LEGAL AUTOPSY

A wide range of samples may need to be taken before, during, or after the gross examination has been completed. The nature of such ancillary investigations will depend upon the nature of death, the history, and the suspicions of the pathologist.

Microbiology

Though more common in clinical autopsies than in forensic work, culture samples for bacteriological, virological, or even mycological study may be needed.

Plain swabs, or swabs immersed in an appropriate transport medium can be employed for sampling a wide variety of sites at autopsy. Tissue samples may be collected in sterile containers, and this is the usual method for virological culture of lung and brain. If blood culture is required to be done, it is best to take blood with a sterile needle and syringe from a large blood vessel such as the femoral vein, before starting the autopsy. Alternatively, blood can be taken from a freshly opened heart chamber using sterile instruments.

In all autopsy investigations, unless cultures are performed soon after death, there is often widespread contamination by organisms that travel rapidly through the dead tissues, especially from the gastrointestinal tract. It requires the expertise of a qualified microbiologist to opine as to what is significant, with reference to the subsequent growth in the laboratory.

Toxicology

Collection of specimens should be done carefully if reliable analytical results are expected. Blood, urine, stomach contents, intestinal contents, organs (liver), CSF, bile, and ocular fluid may be required.[11] The containers in which they are collected must be chemically clean.

The pathologist should submit a form with the samples indicating the nature of analysis required, the personal details of the deceased person, a brief history with details of suspected toxic substances, and a statement as to whether the subject was known to suffer from any infective conditions, including hepatitis or AIDS.

Histology

In most autopsies, the pathologist will need to carry out a histological examination on a range of tissues, even if only to exclude some occult natural disease.

It is customary to retain samples of liver, spleen, kidney, heart, lung, thyroid, adrenal, pancreas, muscle, and brain for this purpose. Where there are indications to examine other parts of the body, these may be retained in addition to the routine tissues.

The required tissue is placed in sufficient quantity of formalin, and allowed to fix for several days before processing. The volume of fixative should be six times the total volume of tissue. There are several specialised procedures in use at present, such as histochemistry, fluorescent microscopy, and immuno-histochemical examination, if evidence of early myocardial infarction is being sought. However, even light microscopy can be helpful in many cases.[12]

Autopsy radiology

Radiology is similar to photography in the autopsy room (*vide infra*), though it requires more bulky apparatus, and some one to operate the equipment and process the films. The quality of radiographic assistance available will vary widely from none to the most sophisticated techniques. In many places,

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radiography may be scarce for living patients let alone corpses, and the same standard of assistance for the pathologist can not be expected.

In more affluent countries, remoteness may be the problem, for which mobile equipment is available and some times used by the forensic pathologist, or the corpse is taken to the radiology department of a nearby hospital for the required x-rays. There are small, portable x-ray kits that can be carried in two suitcases, and can function from an ordinary domestic power supply, or from a portable petro-generator. For isolated organs and tissues, cabinet type radiographic equipment is available, which can be used without the assistance of trained radiographers. In major Forensic Institutes, full radiological facilities with the staff and equipment to operate and process plates is often available.

The usual stage at which radiology is employed is after external examination has been completed, and before dissection has begun. Radiographs to detect bony injury are generally not required, as the skeleton can be inspected directly by dissection. The exception is child abuse, where a full skeletal survey is needed before autopsy.

Suspected gun shot and explosive deaths, air embolism, pneumothorax, and barotrauma should necessarily be subjected to radiological examination before the autopsy, and when a traumatic subarachnoid haemorrhage is suspected, vertebral artery angiography might be necessary.

Mutilated remains from mass disasters also need to be x-rayed, as many victims may have been involved which makes meticulous dissection difficult. Where bombs or explosives are involved, it is necessary to have radiographic facility to detect any parts of the device that may be embedded in the tissues.

Forensic Photography

The type of camera favoured is the 35 mm SLR (single lense reflex), of which there is now a vast variety available in all grades of sophistication and price. Some means of exact focussing is essential (either automatic or split screen), as picture sharpness is of prime importance. The standard 50 mm lens is most useful, but for scenes of crime, a wide-angle lens of 28 or 30 mm is preferable. A longer focal length of up to 80 mm can be useful for close-up pictures of small lesions. Many pathologists prefer to combine lenses into a single variable-focus "Zoom" lens of 28-80 mm range. This saves time spent in changing lenses, and the image resolution is virtually indistinguishable from fixed focal length lenses.

Illumination is usually by electronic flash, an integral part of the camera body. The type of film depends on the nature of illumination. Ultraviolet and infra-red sensitive film has been used to demonstrate surface lesions which are not visible to human eye. It is claimed that occult bruising can be revealed explicitly by ultraviolet photography. Some pathologists use Polaroid cameras either alone or together with conventional film. The advantage of instant prints is that a record of scene of death can be obtained before the autopsy is carried out.

Electronic cameras of varying grades of sophistication, resolution, and cost are now available that can instantly store images on a small internal "floppy disc", which can be viewed immediately on a computer unit, or printed on a colour laser printer. The images can be manipulated in many ways to enlarge sections, or correct colour balance, and can be sent via modems to distant locations, or be incorporated within textual material and reports. All electronic images can be stored on CD ROM equipment, which offers huge storage and retrieval capacities for record keeping and educational

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display purposes. In addition, single frames from video camera recordings can be captured as still pictures and electronically stored.

When photographing viscera the camera should be near enough for the frame to be almost completely filled by the queried object. The shot should be vertical to the lesion, and it is often necessary either to place the dissecting board on the floor, or for the photographer to stand on a stool or some elevation to gain required height.

Where isolated organs are being photographed, they should be placed on a green or blue cloth, such as a discarded operating gown. The organs should be placed on the cloth in one movement and not moved thereafter, otherwise a wet dark stain will mar the green or blue background. The organ should not be oozing blood, and should be dabbed with a dry cloth or sponge just before the photograph is taken to remove shiny wet highlights.

EXHUMATION

Exhumation is the retrieval of a previously buried body for postmortem examination. The term should be restricted to the removal of a body interred in a legitimate fashion in a grave yard (*interment* = burial), rather than the recovery of an uncoffined, clandestinely buried victim of a suspicious death. The latter is actually a "scene of crime," and the pathologist should treat it as such.

Situations requiring a body to be exhumed

- Where some civil legal matter needs to be investigated, such as personal injuries for insurance purpose, or civil litigation relating to negligence - usually after road traffic, industrial, or other accidents.
- Where new information, or substantiated allegations arise to suggest that a death was due to unnatural causes, criminal abortion, or some form of injury or poison, that was not elucidated prior to burial.
- In ancient or historical circumstances, to study either the individual, or a series of individuals, for academic interest.

Procedure

The body is exhumed only on written order from an executive or judicial magistrate. It is done under the direct supervision of the medical officer and in the presence of a police officer. Before beginning to dig, the grave should be positively identified by examining the plan of the grave yard, or by questioning the persons who had accomplished the burial.

The grave is then carefully dug out. In a case of suspected poisoning, about 500 gms of earth in actual contact with the body from the top, sides, and below should be collected and sent for chemical analysis as a control sample, to rule out contamination of the body by a poison present in the soil itself.[13] In coffined bodies, the coffin must be identified by the person who had made it. The coffin or the body is lifted out, and the body is identified by as many persons as possible who had been present at the time of burial. Any debris in the coffin must be sent for chemical analysis.

Exhumation of a recently buried body can be unpleasant, and sometimes hazardous. Disinfectants should not be sprinkled on the body. The examiner should wear masks and thick rubber gloves, and stand away from the direction of the wind. Once the body has been lifted out, postmortem examination is done in the usual way. Viscera should be preserved for chemical analysis. If poisoning by heavy metals is suspected, samples of bone, hair, nails etc., should be preserved. If skeletonisation has taken place, ascertain the age, sex, height, and cause of death.

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Even negative information gained, such as absence of alleged or suspected fractures may be of considerable value.

EXAMINATION OF SKELETAL REMAINS

Skeletal remains sent for examination to a Forensic Expert are usually either those recovered after exhumation, or those recovered from forests, deserted areas, dustbins, ditches, etc. The examination of skeletal remains should be undertaken with great care, in order to provide information which may be vital to the investigation. The following facts should be elucidated.

1. **Human or not :** Can be determined by noting morphological features. It is difficult when only fragments of bone are available. Precipitin test will be helpful in such cases.

2. **Male or female :** In post-pubertal bones, sex differences can be determined from an examination of pelvis, skull, sacrum, etc.

3. **One or more individuals :** This can be ascertained by assorting and assigning the individual bones to their anatomical positions. If there are no disproportionate or duplicated bones, and the age and sex of all the bones are same, it can be safely concluded that all bones belong to the same individual.

4. **Probable stature :** Can be assessed by measuring long bones and applying appropriate formulae.

5. **Age of the individual :** Can be determined by the examination of teeth, ossification status, and suture closure.

6. **Identity :** The superimposition technique can be employed for establishing identity. If tooth pulp or bone marrow is available, DNA fingerprinting can be done.

7. **Time of death :** It is difficult to tell precisely the time of death. But an intelligent guess may be made from the condition of the soft parts, ligaments, and bones. Skeletonisation generally takes place within three to six months after burial, and sooner if the body is exposed to inclemental conditions.

8. **Cause of death :** It is usually impossible to find out the cause of death from an examination of bones, unless there is evidence of major fractures in the vital parts of the skeleton (e.g., skull). However, it is often difficult to distinguish between an antemortem fracture and a postmortem artefact. Presence of blood clots, or evidence of reparative process at the fracture site indicates antemortem nature.

POSTMORTEM ARTEFACTS

Forensic Medicine is best learned by a judicious combination of theoretical and practical knowledge. A good Forensic Expert is one who has not merely a vast experience in conducting autopsies, but one who has trained himself to make precise and correct interpretation of findings. One must not allow

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Box 7.1 Postmortem Artefacts

- Aspiration of gastric contents may occur as an agonal terminal event, or due to resuscitation, or handling of the body after death. This should not be confused with death due to choking.
- Defibrillator used as a part of resuscitative procedure may produce contusions over chest. Similar is the case with external cardiac massage which may even cause ribs to be fractured.
- Positive pressure breathing done through a respirator may cause emphysematous condition of the lungs.
- Fractures of skull, cervical spine (*undertaker's fracture*), ribs, or extremities may result from rough handling of a dead body.
- Abrasions may result from a recently dead victim being dragged on a rough surface.
- Rough handling can break rigor mortis leading to wrong conclusions about the time of death.
- Bodies placed in cold storage immediately after death can develop pink colouration of postmortem lividity, which may be mistaken for carbon monoxide poisoning.
- Postmortem lividity in internal organs such as the heart, stomach, intestines, etc., may be mistaken for congestion, inflammation, or disease.
- Tight clothes can produce marks resembling ligature mark (neck), or bruising (trunk).
- In a burnt body, fractues of skull or limb bones can occur due to heat, and must not be confused with traumatic fractures.
- Insect or animal bites on a dead body may be mistaken for antemortem injuries.
- Decomposition can cause a number of changes that may be mistaken to be due to antemortem violence, e.g., oozing of blood stained fluid from mouth and nose.

Box 7.2 The Disposal of the Dead

- **Christian** - Body is laid out after washing, and clothed in a shroud or night attire. Roman Catholic faith prefers a crucifix to be laid on the chest of the deceased, or that the arms should be folded as a cross.
- **Muslim** - Body is washed by a professional, after the head has been turned towards Mecca. It is then clothed in 3 pieces of white cloth 9 yards long, covering the upper, middle, and lower portions of the body. Generally, no coffin is used.
- **Hindu & Buddhist** - Body is washed, wrapped in a shroud, covered with flowers, & cremated.
- **Jewish** - Body is laid out with the eyes closed, and the feet pointing towards the door. A purification ritual called "Tahara" is performed by 2 specially qualified Jews, and the body is then buried wrapped in a shroud.

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dogmatism or inflexibility to cloud one's judgement.[14] A self-opinionated expert is a poor expert.

There are several inherent pitfalls that must be avoided in the course of medicolegal autopsies which can lead to erroreous or fallacious conclusions. Every forensic pathologist must familiarise himself with these *postmortem artefacts* that are liable to misinterpretation. While an exhaustive discussion on this matter is unfortunately beyond the scope of this book, some of the more commonly encountered artefacts have been mentioned in **Box 7.1**

NEGATIVE or OBSCURE AUTOPSY

When at the conclusion of a complete and thorough postmortem examination, inclusive of all relevant investigations such as histopathological, toxicological, and biochemical investigations, no cause can be attributed to the death, it is termed as a **negative autopsy**. Approximately 2 to 5% of all autopsies are negative in nature.

Causes

1. Inadequate history.
2. Natural disease which is difficult to establish as a cause at autopsy, e.g., cardiac arrhythmia, uraemia, adrenal insufficiency, etc.
3. Death due to vagal inhibition.
4. Death due to concussion.
5. Death due to anaphylaxis.
6. Death due to certain kinds of poisons, e.g., anaesthetics, snake bite, etc.
7. Electrocution deaths.

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CHAPTER - 8

Mechanical Injuries

"He jests at scars that never felt a wound".
- Shakespeare, Romeo and Juliet, II, 2.

Introduction

Trauma is an insult to the state of well being. This insult can be physical or mental. Usually, one is accompanied or followed by the other in negligible or substantial degree. Physical trauma may manifest itself as a functional change, or structural (anatomical) change, or both. The kind of physical trauma which causes structural (anatomical) change is called an **injury**. Injury may be associated with loss of tissue, or without recognisable loss (e.g., redness, oedema). An injury which is associated with loss of tissue is called a **wound**. In other words, all wounds are injuries, but all injuries are not wounds. However, injury and wound are often described as being synonymous. For the sake of convenience, the term injury will be used in place of wound in this textbook even though it may not be accurate, since it enjoys wider acceptance.

Injuries can occur due to natural causes (e.g., diabetic ulcer), or due to unnatural causes which may be *physical* (e.g., heat, cold, electricity, radiation, etc), *chemical* (e.g.,corrosives), or *mechanical* (e.g., weapons such as knife, stick, etc). A **mechanical injury** can be adequately defined as "Damage to any part of the body due to the application of mechanical force". The damage referred to is loss of tissue.

CLASSIFICATION OF MECHANICAL INJURIES

- Abrasion
- Contusion
- Laceration
- Incision
- Puncture (stab)
- Fracture

Abrasion

An abrasion is the loss of superficial layers of skin or mucous membrane due to mechanical force. Because of the corrugated nature of dermoid papillae, abrasions usually involve the corium of dermis. Since the blood vessels are confined to dermis, such involvement of corium seems to be the reason for the common occurrence of bleeding in abrasions.[1] There is also oozing of lymph, which dries up after death making the abrasion firm, leathery, or parchment-like.

Mechanism of occurrence : The mechanical force which causes an abrasion is either *friction* (sliding), or *pressure* (compression) applied on the body surface. Usually both these elements are present, with one predominating over the other. Friction-predominant abrasions are called **tangential abrasions**, while those which are compression-predominant are referred to as **pressure abrasions**.

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Tangential Abrasion: A tangential (frictional) abrasion is referred to as a **linear abrasion** or **scratch** if it is in the form of a linear mark caused by a narrow, sharp object. A wider mark caused by a broad, rough surface is a **brush abrasion** or **graze**.

i. Linear abrasion (scratch) (**Fig 8.1**): This is produced by frictional (sliding) movement of a sharp object such as a finger nail, pin, thorn, etc. The object causing the linear abrasion carries the torn superficial layers of skin in front of it as it slides forward. This heaping up of superficial layers, often visible at the concluding end of a linear abrasion, is an indicator of the direction of movement of the causative agent.

ii. Brush abrasion (graze) (**Fig 8.2**): This is produced when a rough object scrapes over the skin, or when the skin moves against a broad, rough surface (e.g., fall on a road). The terminal end of injury often shows a serrated border with piling of torn layers of skin, again giving a fair indication of the direction of sliding movement.

Pressure Abrasion: This is caused by pressure of an object on the skin. When the impact is vertical, the epidermis gets crushed, and an imprint of the impacting object is stamped upon the surface. Such an injury produces a replica of the patterned surface of the causative object, and is called an **impact abrasion** (e.g., radiator grill impression on the skin of a victim of vehicular impact). In the absence of an impacting force, if there is merely sustained pressure, the resulting abrasion can still reflect the pattern of the object, and is referred to as a **contact abrasion** (e.g., ligature mark of hanging or strangulation). In such **patterned abrasions** (**Fig 8.3**), the pressure of the object is mainly vertical, with the frictional (tangential) force being negligible. As the element of frictional force gradually increases, the abrasions begin to fail to exhibit the weapon pattern and are referred to as **non-patterned abrasions.** Depending on the presence and extent of crushing, a contused area can surround these abrasions to such an extent that in some cases, the contused area will be more marked than the abrasion, which is then called an *abraded contusion*. Alternatively, if the abraded part is more prominent, it is more appropriately called a *contused abrasion* (**Fig 8.4**).

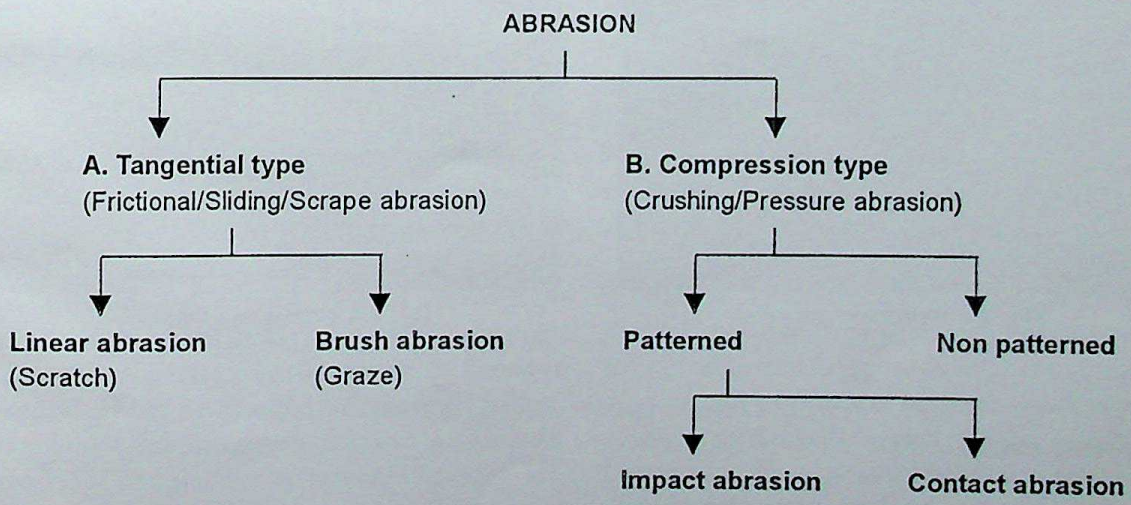

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Fig. 8.1 Linear abrasion

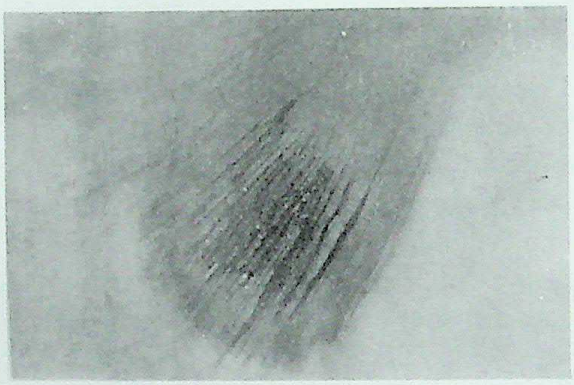

Fig. 8.2 Grazed abrasion

Fig. 8.3 Patterned abrasion

Fig. 8.4 Contused abrasion

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Other Abrasions : Abrasions can be induced after death. They are called **postmortem abrasions**. They are quite common, and can occur due to mechanical force (e.g., dragging the body after death) or by animal action (e.g., ant bite). Even the normal procedures of post-autopsy reconstruction and handling in the mortuary may cause dermal changes simulating abrasions, especially after washing with hot water. While an abrasion caused during life is covered by scab due to coagulation of blood and lymph, and is associated with signs of inflammation, a postmortem abrasion does not demonstrate these features. In fact it usually appears pale and dry. Ant bite marks are pale and irregular in shape, and are seen mainly in moist regions such as the areas around the eyes, nose, mouth, genitalia, and axilla. Abrasions produced immediately before or after death may be difficult to differentiate even by microscopic examination.[2]

An abrasion fabricated on the body by oneself, or with the help of another person, is called **fabricated abrasion**. In the former case, only accessible parts of the body are involved such as face, hands, etc. The motive for such fabrication can be serious or trivial.

Repair and healing : Healing involves two phenomena - contraction of the wound, and replacement of lost tissues.[3] When fresh, an abrasion is bright red in colour. Scab forms by 12 to 24 hours. When fully formed, a scab is red in colour, and is moist, but it becomes reddish brown and dry by about 2-8 days. Abrasions heal from the periphery by new growth of epithelial cells. Ordinarily the scab falls off by about 7-8 days, leaving a pale area which may persist for a few days or weeks (**Fig 8.5**). A rough estimate of the age of an abrasion can thus be made from these features. Earlier stages (before scab formation) can be assessed by histology. Infiltration of polymorphonuclear cells in a perivascular formation signifies that the wound is 4-6 hours old. At 12 hours, the wound shows three layers - a surface zone of fibrin and red cells, a deeper zone of polymorphonuclear cells, and a third layer of damaged, abnormally staining collagen.

Medicolegal significance: An abrasion indicates the site of contact with an object. Abrasions are normally simple wounds. They are actually three dimensional, but for practical purposes they are considered to be two dimensional. The type of abrasion may reveal as to whether the manner of contact was a sliding one or compressing one. The pattern (if present), can help to some extent in deducing the nature of the object, while the stage of healing process could help in determining the time of occurrence. However, the determination of age of an abrasion is usually only approximate and is seldom accurate.

The exact site of an abrasion can sometimes give valid information about the nature of crime (e.g., abrasions around the nose and mouth suggest smothering). It is advisable to look for foreign material over abrasions (e.g., presence of mud particles indicates a fall in a muddy area).

Contusion

A contusion is an extravascular collection of blood that has leaked from blood vessels damaged by mechanical impact of a blunt nature, without loss of continuity of tissues. A contusion is also referred to as a **bruise**. The blood vessels involved are usually small arteries, veins, or capillaries. Contusions are usually situated in the subcutaneous tissue, but can also occur in various internal viscera.

Mechanism of occurrence : The extent of effusion of blood resulting from damage to blood vessels depends on several factors such as the *site* of wound (effusion will be greater in lax tissues such as periorbital area), *thickness* of skin (effusion is lesser if the dermis is thick, as in the palms and soles), *resilience* of the area (effusion is minimal in the well

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Fig. 8.5 Healed abrasion

Fig. 8.6 Ectopic bruise

Fig. 8.7 Linear bruises showing "Tram line pattern" caused by beating with a stick

Fig. 8.8 Postmortem contusion

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yielding anterior abdominal wall), *sex* (females contuse more easily than males due to higher amount of adipose tissue), and *age* (children and aged individuals are more susceptible than young adults). Elderly people bruise easily due to increased fragility of blood vessels. The degree of effusion is also directly proportional to the vascularity of the area, and the amount of force applied. Diseases such as haemophilia, scurvy, and leukaemia can aggravate effusion. A bruise will be less prominent if it is situated in deeper tissues.

Blood escaping from ruptured vessels usually percolates along the lines of least resistance. Hence, it is easily influenced by gravity. The site of a contusion therefore does not necessarily indicate the site of application of force, e.g., black eye in head injury. A "black eye" can be caused by a direct injury, or by gravitation of blood from a wounded scalp, or from a fractured orbit. Contusions appearing at a site remote from the site of application of force are called **percolated contusions** or **ectopic bruises** (Fig 8.6). A deeply situated bruise may manifest externally only after several hours or days. Such a bruise is called a "**come-out bruise**." This may be due to haemolysis, where-in the freed haemoglobin stains the tissues more and more densely as time elapses.

On certain occasions, the shape of a bruise may give valuable information about the causative object. Bruises caused by finger tips over the neck in throttling represent one such example. A lash by a whip produces a bruise in the form of double lines with an intervening space approximately equal to the width of the whip. Such bruises are called **patterned bruises** (Fig 8.7).

The size of a contusion may roughly correspond to the object causing it. This gives some clue regarding the weapon employed. Thus a contusion caused by a stick is longer than it is broad. Its width usually corresponds to the diameter of the stick. With lapse of time however, the contusion may appear larger in size than it originally was, due to continued extravasation of blood and development of oedema.

Repair and healing : After a lapse of some time, the red blood corpuscles in a bruise will begin to disintegrate by haemolysis, liberating haemoglobin, which in turn gets broken down into haemosiderin, haematoidin and bilirubin, by tissue enzymes and histiocytes. These pigments impart certain colour changes to a bruise which can be visualised, especially in the case of fair complexioned individuals. To begin with, the injury is red in colour, turning bluish in a day, and brownish over the next 3 days due to haemosiderin. It turns greenish over the subsequent 3 days (haematoidin), and yellowish in the final 2 days (bilirubin). At the end of a fortnight, the original normal colour of the skin is restored, since these pigments get removed by phagocytosis. In subconjunctival haemorrhage, due to diffusion of atmospheric oxygen, these colour changes may not be noticeable.

Postmortem lividity (hypostasis) in the early stages can mimic a contusion, posing problems in medicolegal interpretation. The important differentiating points are summarised in **Table 8.1**.

Contusions can be produced after death, and are referred to as **postmortem contusions** (Fig 8.8). This is particularly likely when moderate force is applied over those areas where the skin is immediately subjacent to underlying bone (e.g., forehead). A severe blow to the body within a few hours of death can produce contusions in other parts of the body also. However such contusions are devoid of swelling, and do not demonstrate inflammatory reaction. Their edges appear sharply demarcated.

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Table 8.1 Differences Between Bruise And Postmortem Lividity

	Bruise	Postmortem lividity
1. Cause	Rupture of vessels and extravasation of blood	Engorgement of vessels due to pooling of blood
2. Site	Anywhere in the body	Only in dependent parts
3. Surface	Elevated	No elevation
4. Colour	Variable, depending on the age	Normally purple
5. Incision	Shows extravasated blood in the cut tissues which cannot be washed off.	Bloos oozes out of cut vessels which can be easily washed off
6. Histology	Signs of inflammation present	No signs of inflammation

Medicolegal significance: A contusion need not necessarily indicate the exact site of contact with a blunt weapon, since it can appear at an area remote to the application of force. The size of a contusion may be smaller or greater than the weapon producing it. However, it can give some clue about the contact of a body with an object, and depending on the site of occurrence, to the nature of crime (e.g., contusions in and around the nose and lips suggest smothering). In several cases, the nature of the object used, and the degree of force applied can be reasonably presumed. Colour changes can help to deduce the time of occurrence. In ordinary circumstances a contusion is a simple injury, unless it is multiple, large, or involves vital internal organs. Contusions are three dimensional, but in a living victim, the depth cannot normally be measured.

Laceration

It is a type of mechanical injury where there is a rupture or tear of skin or deeper tissues, or both, due to application of blunt force. Lacerated injuries are three dimensional with length, width and depth, and often have abraded margins, and irregular, contused edges. Since blood vessels are crushed, bleeding is usually not severe. However, substantial bleeding can occur if blood vessels get torn partially. Foreign particles derived from the weapon or impacting surface are frequently present in the wound. The interior of the wound contains bridging strands of tissue which can be visualised clearly with a hand lens. The depth may not be uniform throughout the injury.

Classification: Lacerated injuries can occur from splitting, stretching, grinding, or compression. Accordingly they are classified as:

- Split laceration (incised-looking laceration)
- Stretch laceration
- Grind laceration (avulsion)
- Cut laceration

Split Laceration (Fig 8.9): In this type, the skin gets split due to compression between the weapon and underlying bone. The edges often appear clean cut on cursory inspection. Hence these injuries are also called **incised-looking lacerations**. However, a hand-lens examination will reveal contusion of edges and bridging of tissues. Common areas of involvement include scalp, lower jaw, shin, etc.

Stretch Laceration (Fig 8.10): Here the skin gets stretched to a breaking point resulting in a laceration at that site. For example, pressure over the thigh which stretches the skin towards the knee can cause a laceration along the inguinal line. Ordinarily,

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Fig. 8.9 Split laceration

Fig. 8.10 Stretch laceration

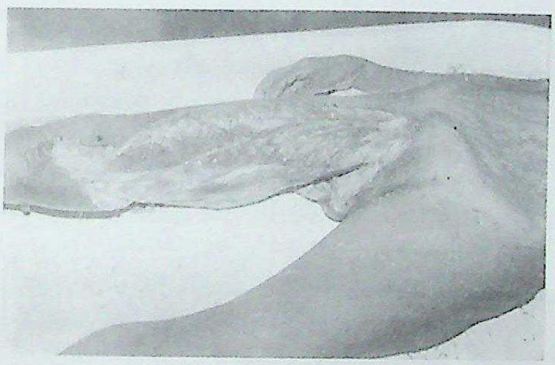

Fig. 8.11 Avulsed laceration

Fig. 8.12 Incised wounds

Fig. 8.14 Chop wound

Fig. 8.15 Multiple stab wounds

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unless great force is used, most lacerations require a firm base to act as an anvil for the skin and underlying tissues to be pinned against. But this is not true for a stretch laceration.

Grind Laceration (Avulsion) (Fig 8.11): In this type, there occurs a separation of skin from underlying tissues due to the shearing or grinding action of a weight, for e.g., the wheel of a heavy vehicle.

Cut Laceration: This type of laceration results from the cutting action of a weapon which is not very sharp, for e.g., a broken glass piece. It is a lacerated wound with profound and distinct contusion of edges. While incised wounds divide hairs cleanly, lacerations cause some hairs to be driven into the interior.

Other Types: 1.**Patterned laceration** - Lacerations usually do not reproduce the shape of the injuring object. However, rarely the shape is recognisable, as in the case of a hammer blow to the scalp. But, calculation of the exact size of striking surface in such cases can be misleading. 2.**Postmortem laceration** – Lacerated injuries may be produced by terrestrial or aquatic animal action (rat, fish, crab, etc.) on corpses. Absence of vital reaction will assist in distinguishing them from antemortem lacerations. The edges may also show characteristic evidence of 'nibbling' or 'gnawing'. A running vehicle or train can cause extensive postmortem lacerations of a dead body lying in its path.

Repair and healing: Lacerated wounds often get infected, thus delaying the healing process. As they do not exhibit uniform healing pattern, age estimation is difficult and unreliable. Healing is by second intention. It begins as phagocytosis to remove debris, followed by filling of the defect by granulation tissue. Specialised tissues lost are repaired, and epithelium regenerated to cover the surface. A permanent scar is therefore inevitable. The time frame will depend upon the extent and severity of the laceration.[4]

Medicolegal significance: Lacerations are usually either accidental or homicidal. Self inflicted lacerations are rare. Foreign particles in these wounds may help to identify the causative object. The exact type of lacerated injury may provide a clue to the nature of occurrence. In the case of avulsions, the direction of force can often be ascertained. The site of the injury may provide some clue about the cause of death, for e.g., lacerations of internal mucosa of lips may suggest smothering. Lacerated wounds of face constitute grievous hurt because of permanent disfiguration.

Incision

An incision or incised wound is a clean-cut separation of skin or deeper tissues (or both), caused by the sharp cutting edge of an object, without loss of substance (Fig 8.12). The commencement of the wound is usually deep, gradually becoming shallower, and finally tailing off towards the end. This tailing of the wound indicates the site of withdrawal of the weapon from the body, suggesting the direction in which the wound was inflicted. The wound is generally straight and shows maximum retraction of the edges at the centre. In other words, the wound "gapes", because of which the width of the wound is greater than the width of the weapon causing it. The gaping is more if the incision is made at right angles to the "lines of cleavage" (*Lines of Langer*), and much less if it is parallel to these lines,[5] (Fig 8.13).The edges of the wound are clean cut and may be everted. Ordinarily there is no bruising of the margins. An incised wound is necessarily longer than it is deep. It bleeds freely, as blood vessels are cleanly cut and not crushed.

Classification : An incision produced by a light cutting weapon is smooth with minimal bruising of margins. Heavy cutting weapons such as an axe or cleaver produce distinct contusion of the edges, and the resulting injury is sometimes referred to as a **chop wound** (Fig 8.14). Incised wounds produced

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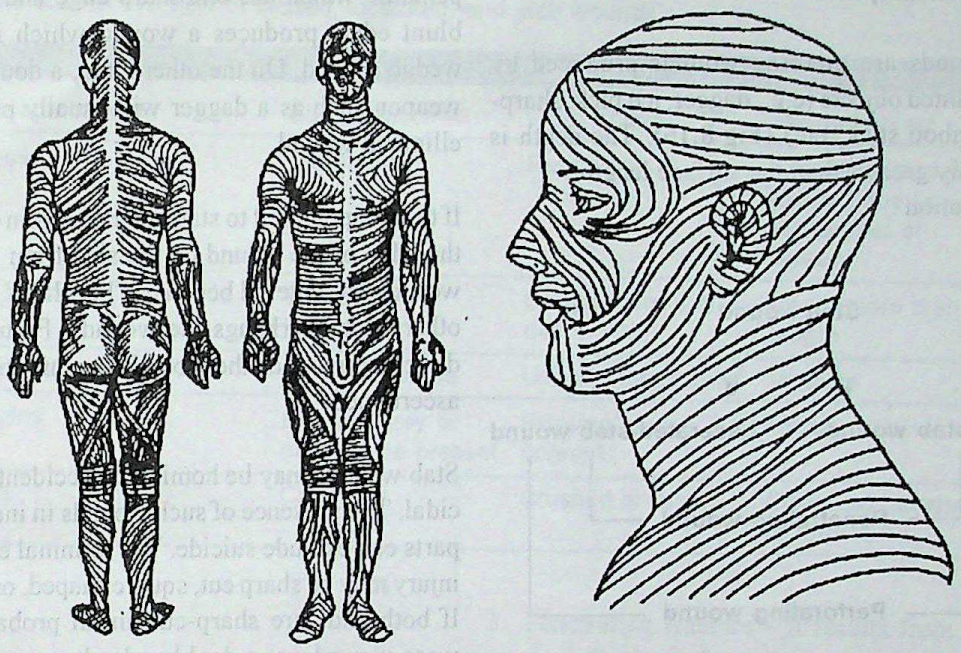

Fig. 8.13 Lines of Langer

over wrinkled skin may appear as lacerations, due to irregularity of edges, e.g., in the scrotum. A hand lens examination will help to differentiate them as incised wounds. Such wounds are sometimes called **lacerated-looking incised wounds.**

Repair and healing : In the case of an incised wound with very little damage to tissues on either side of the cut, healing occurs without delay, if the edges are apposed properly. Small blood vessels which are cut get occluded by thrombosis. Fibrin precipitated locally will then link the two edges. Coagulated blood on the surface forms a scab and helps to keep the wound clean. This occurs within 12-24 hours. Over the next few days, capillaries proliferate to bridge the gap, and fibroblasts secrete collagen. In 3-5 days, vessels are thickened, and many of them get obliterated, which is followed by scar formation. This is usually complete by about the 6th day. The scab over the wound then falls off, leaving behind a soft, tender, reddish scar, which in the course of weeks or months becomes whitish and firm. Infection can however delay the healing process.

Medicolegal significance : Incised wounds invariably point to the use of a cutting weapon, (light or heavy). The length, width, or depth of an incised wound usually do not correspond to the length, thickness or width of the cutting object. Tailing gives an indication of the direction of movement of the cutting edge.[6] Also, the site may suggest the possible motive, intent, or even the mental status of the assailant. For e.g., incised wounds concentrated over the genital area reveal a sex-related motive. Homicidal incised wounds sometimes suggest the disturbed mind of the assailant, which may vary from cold fury to irrational psychosis.

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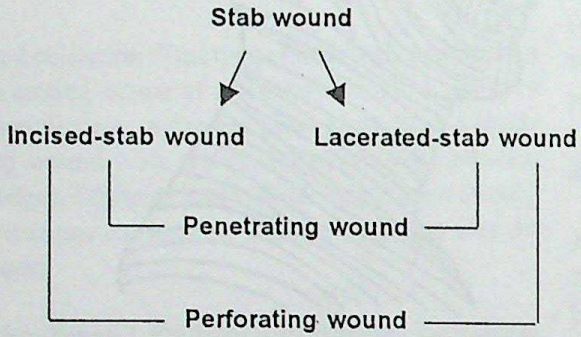
Stab (Puncture)

Stab wounds are piercing wounds produced by sharp-pointed objects (e.g., dagger, ice pick, sharpened bamboo stick, etc) (Fig 8.15). The depth is necessarily greater than the surface length.

Classification :



Stab wounds are divided into **penetrating wounds**, if they *terminate* in tissues, organ, or cavity, and **perforating wounds**, if they *transfix* them (through-and-through wounds). In the former, there is only one surface wound, while in the latter, there will be two separate surface wounds, one caused by the *entry* and the other by the *exit*. External bleeding may be slight, but internal bleeding can be very profuse.

In perforating wounds, the wound of entry is usually larger than the wound of exit, because weapons used for stabbing usually taper towards the tip. The edges of the wound of entry are inverted, while they are everted in the wound of exit. Clothes may be pushed into the wound of entry. The direction in which the wound has been inflicted can be deduced by joining the wounds of entry and exit.

Medicolegal significance: The external appearance of a stab wound may sometimes give a clue regarding the kind of weapon used in inflicting it. Thus, a penknife which has one sharp edge and a thicker, blunt edge, produces a wound which is usually wedge shaped. On the other hand, a double-edged weapon such as a dagger will usually produce an elliptical wound.

If the weapon used to stab enters the skin obliquely, the edge of the wound on the side from which the weapon has entered becomes "bevelled", while the other edge overhangs the wound. From this, the direction in which the wound was inflicted may be ascertained.

Stab wounds may be homicidal, accidental, or suicidal. The presence of such wounds in inaccessible parts can exclude suicide. The terminal ends of the injury may be sharp cut, square shaped, or rounded. If both ends are sharp-cut, in all probability the weapon used was a double-edged one, even though a single-edged weapon can sometimes produce such an appearance due to splitting, or if the weapon possesses a curved beak. It can also occur if a single-edged weapon is tilted while being withdrawn. Bruising around the stab wound may indicate that the weapon had been inserted up to the hilt.[7]

The depth of the wound may give an indication of the minimum length of the weapon used, provided the entry wound and the termination of the wound track are situated in non-yielding areas. If the punctured wound has made an entry through a yielding site (e.g., abdominal wall), or ends up in a cavity or a collapsible organ (e.g., lung), the deduction of depth becomes a matter of conjecture. The length of the entry wound may be equal to the breadth of the stabbing weapon. However, it is usually lesser by 0.2 to 0.3 cm if the skin is elastic, while it may be greater if rocking of the object occurs during the course of stabbing.[8] The width of the entry wound is usually greater than the thickness of the weapon due to gaping.

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Table 8.2 An overview of incised, lacerated, and stab wounds

	Incision	Laceration	Stab
Causative agent	Sharp object	Blunt object	Pointed object
Edges	Clean-cut	Ragged	Clean-cut, and often everted due to withdrawal of weapon
Dimensions	Length more than depth	Length more than depth	Depth more than length
Haemorrhage	Usually profuse	Usually not profuse	Variable
Margins	Bruising may or may not be present	Bruising always present	Usually absent
Hairs	Clean-cut	Crushed or torn	Clean-cut, crushed, or torn

Fracture

Fracture of a bone is defined as breakage of a bone due to direct or indirect force.

Classification[9]:

***Direct Fractures*:**

1. Focal fracture - This is otherwise known as **tapping fracture** and results from a small force applied to a small area. It is usually transverse. Injury to overlying soft tissues is minimal. Typically seen in regions such as the forearm or leg where two bones lie adjacent to each other, only one of the two bones being fractured, for e g., while warding off blows during an attack.

2. Crush fracture - It results from application of a large force over a large area, and is typically *comminuted* in type, i.e., fragmented. Injury to surrounding soft tissues is usually extensive. If two bones lie adjacent to each other, both are involved, for e.g., bumper fracture of the tibia and fibula in a pedestrian who has been hit by a car.

3. Penetrating fracture - It results from application of a large force over a small area. Bullet injury to a bone is a classical example of this type of fracture.

***Indirect Fractures* (Fig 8.16):**

1. Traction fracture - It results when a bone is pulled apart by traction, e.g., transverse patellar fracture due to violent contraction of quadriceps.

2. Angulation fracture - It occurs due to bending of a bone. The concave surface of the bend is compressed, while the convex surface is put under traction resulting in breakage.

3. Rotational fracture - Here the bone is twisted to produce a spiral fracture.

4. Vertical compression fracture - In this type, there is an oblique fracture of a long bone such that the hard shaft of the bone is driven into the cancellous portion. It is sometimes encountered in the distal femur of a vehicle passenger when

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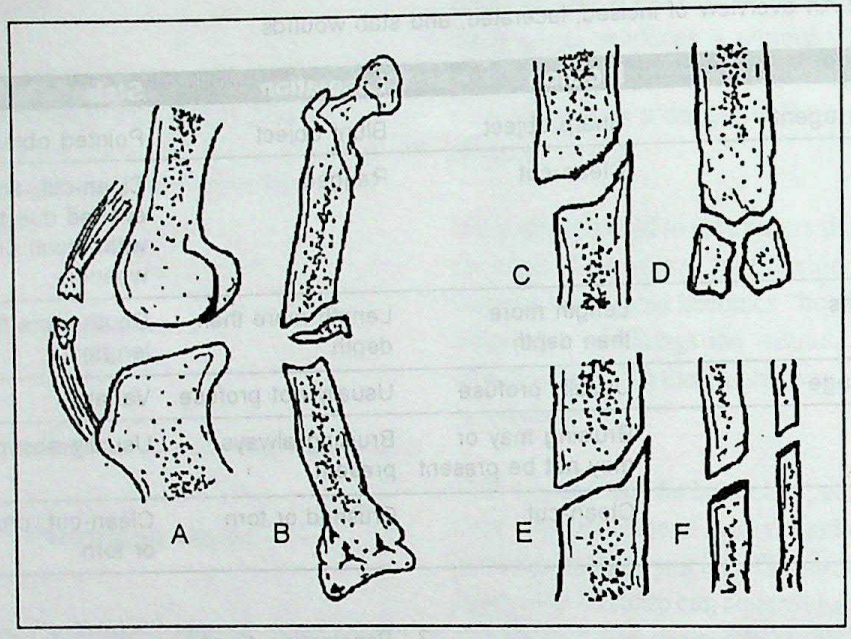

Fig. 8.16 Indirect fractures.
A - Traction, B - Angulation, C - Rotational, D - Vertical compression, E & F - Angulation compression

there has been a collision, and his knee has impacted violently against the dashboard.

5. Angulation-compression fracture - Here, the fracture line is curved, with an *oblique* component due to compression, and a *transverse* component due to angulation.

Repair and healing : Healing of a fracture depends upon the age and nutritional status of the individual. In general, cancellous bone unites faster than cortical bone. There are 5 stages involved in the healing process:-

- Haemorrhagic phase
- Proliferation phase
- Callus phase
- Consolidation phase
- Remodelling phase

In the *haemorrhagic phase*, there is haemorrhage at the site of fracture. In the *proliferative phase*, a collar is formed around the fractured ends by proliferation of cells from periosteum and endosteum. In the *callus phase*, cellular elements give rise to osteoblasts and chondroblasts which produce a matrix of collagen and polysaccharide, impregnated with calcium. This is called "callus." In children, the callus is visible radiologically within 2 weeks of the fracture. In the *consolidation phase*, the callus is transformed into mature bone which takes about 4-6 weeks in children, and approximately 3 months in adults. In the final *remodelling phase*, the mature bone is remodelled.

Medicolegal significance: Fracture of a bone, however trivial, constitutes grievous hurt according to law. From a study of the exact type of fracture an idea about the causative force can be estimated, i.e., whether the force was direct or indirect, rotational or angular, etc. The site of fracture may help to indicate the cause of death, for e.g., fracture of hyoid bone suggests throttling. The time of occurrence of a fracture can be estimated roughly from the stage of the healing process.

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CHAPTER - 9

Firearm Injuries

"A cartridge case at the scene of offence could prove as incriminating as if the murderer had left his visiting card".

- Sir Sydney Smith

Introduction

Ballistics

Ballistics is the science dealing with the study of firearms and ammunition. It includes the study of the effects of discharging a firearm, projectile motion, and factors affecting the efficacy of firing. While a detailed knowledge of these is not essential for a forensic pathologist, he must nevertheless acquire an elementary knowledge of firearms for proper interpretation of the wounds caused by them.

Sub-Divisions[1]

1. Proximal (Internal) Ballistics: The study of firearms and projectiles is called proximal ballistics.
2. Intermediate (External) Ballistics: The study of the motion of a projectile after being ejected from the firearm till the time it hits the target is termed as intermediate ballistics.
3. Terminal (Wound) Ballistics : The study of injuries produced by firearms is referred to as terminal ballistics, and is the exclusive realm of a forensic pathologist.

Proximal Ballistics

In simple terms, a **firearm** is a mechanical device equipped to create an explosion, which in turn forces out a projectile at high velocity in order to hit a target. The term **ammunition** refers to the materials used for causing the explosion, along with the projectile. One complete round of ammunition is called a *cartridge*. The projectile is pushed out through a cylindrical tube which is called a *barrel*. If the barrel is grooved internally, it is a *rifled* firearm, otherwise it is a *smooth bore* firearm.

CARTRIDGE

A cartridge consists of an outer *case* in which the explosive is stored together with the chemicals required to ignite it, and the projectile. The explosive is called *propellant* or *gun powder*, while the igniting chemical is called *detonator* or *primer*. The projectile is either a *bullet* or *pellets* (*shot*). The former is used in **rifled firearms**, and the latter in **smooth-bore firearms**.

Cartridge =			
Case +	Detonator + (*primer*)	Propellant + (*gun powder*)	Projectile (missile) (*Bullet* or *shot*)

i) Cartridge case (**Fig 9.1**): In rifled firearms, the cartridge case is made of an alloy of copper and zinc, while in smooth bore firearms it is made of a special type of paper or card board. In both

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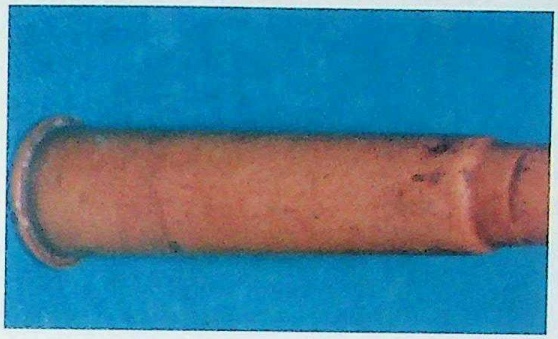

Fig. 9.1 Cartridge case a) Rifle

Fig. 9.1 b) Shot gun

Fig. 9.2a Rim

Fig. 9.2b Groove

Fig. 9.3 Projectiles a) Bullet

Fig. 9.3 b) Pellets

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types the shape is cylindrical. One end of the case is closed with a plate of brass and is called the base, while the other end is open. The cartridge case helps not only to keep the contents in place, but also provides a water-proof cover for them and protection against deterioration. The base of the cartridge is invariably rimmed, but in automatic weapons there is a groove instead of the usual projecting rim, (**Fig 9.2**).

ii) Detonator : It is also known as "primer". Common ingredients comprise mercury fulminate (or lead azide), potassium chlorate, and antimony sulfide. These chemicals are capable of rapid combustion. They are stored in a small compartment attached to the base of the cartridge case called *percussion cap* which is made of copper. Hence the detonator is also known as *cap composition*. In most of the cartridges the percussion cap is situated near the centre of the base, and are therefore referred to as *centre fire* cartridges, while in a few the whole of the base acts as a percussion cap and are known as *rim fire* cartridges.

iii) Propellant : This is situated in close proximity to the primer within the cartridge case. There are three types of gun powder - *black powder*, *semi-smokeless powder*, and *smokeless powder*.[2] Black powder is a mixture of 75% potassium nitrate, 15% charcoal and 10% sulphur. Smokeless powder consists of nitrocellulose alone (*single base*), or nitrocellulose combined with nitroglycerine (*double base*). Semi-smokeless powder consists of 20% smokeless powder and 80% black powder. While all types of gun powder produce smoke, the smokeless variety does not produce as much smoke as black powder, and is the most effective type being capable of imparting higher velocity to the projectile. The ignited primer causes combustion of gun powder by which hot gases under tremendous pressure are produced within the closed confines of the cartridge, propelling out the projectile with great velocity. A single grain of black powder can produce 200 to 250 cc of gas composed of carbon monoxide, nitrogen, hydrogen, hydrogen sulphide, and traces of methane and oxygen, while a grain of smokeless powder can generate 800 to 900 cc of gas.

iv) Projectile : This is usually either a bullet or a bunch of pellets (shot), (**Fig 9.3**). The former is used in rifled firearms, while the latter is used in smooth-bore weapons. Firearms meant to propel projectiles of less than one inch diameter are called *small arms*. They may be hand operated, i.e., *hand arms* (e.g., pistol and revolver), or they may be placed against the shoulder and fired, i.e., *shoulder arms* (e.g., rifle, musket, shot gun, etc).[3] Hand arms are also referred to as *side arms*. In *country-made firearms*, unusual projectiles are often used, e.g., pieces of glass, cork, wood, nails, stones, etc.

Normally, a bullet is held in the cartridge case by a groove called *cannelure*. As a result of the pressure generated by gases, the bullet gets released, and is pushed through the barrel and out of the muzzle with great velocity. Bullets are usually made of lead with a coating of cupronickel alloy. They are invariably conical in shape, in order to offer least resistance while travelling through air. The tip of a bullet is referred to as its *nose*. Depending upon the size of the bullet, it can be short, medium or large, and as per the shape of the nose, it can be flat nosed, round nosed, hollow nosed or point nosed. Pellets used in smooth bore firearms are collectively referred to as *shot*. Sometimes *slugs* may be used instead, which are much larger. A flat felt-disc or compressed paper or cardboard called *wad* lies between the pellets and propellant. It is often called *underwad*. There is another wad, termed the *turnover wad* or *top wad*, situated over the lead shot, the purpose of which

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is to keep the shot in place. Wads not only hold the pellets and gun powder tightly in place, they also act as a piston sealing the bore effectively, thus preventing the expanding gases from escaping. This is known as *obturation* or *sealing* of the bore which is of vital importance. In order to lubricate the bore, wads are often impregnated with grease. In modern ammunition, plastic is being used increasingly commonly, and some of the older constituents are no longer present.[4]

Once a firearm has been discharged, the empty cartridge case is left behind in the chamber devoid of projectile, but with residues of primer and detonator still present. This is called *spent cartridge*. The spent cartridge has to be extracted from the chamber manually, or it may get ejected automatically (as in automatic firearms).

Figs 9.4 and 9.5 show the longitudinal section of shotgun and rifled firearm cartridge respectively.

FIREARM

Every firearm has a *chamber* in which the cartridge is normally lodged. The chamber leads into a hollow steel tube called the *barrel*. The projectile released from the cartridge in the chamber is forcefully pushed through the barrel by the expanding gases produced by the explosion of gun powder. The proximal end of the chamber has a minute hole, through which a *pin* moves forward to strike the base of the cartridge.This striking action produces heat which ignites the primer. This pin, known as *firing pin* is activated by a spring connected to a trigger mechanism. The *spring* is released on pulling the *trigger*. The part which incorporates the firing pin, spring, and trigger is called the *bolt*.

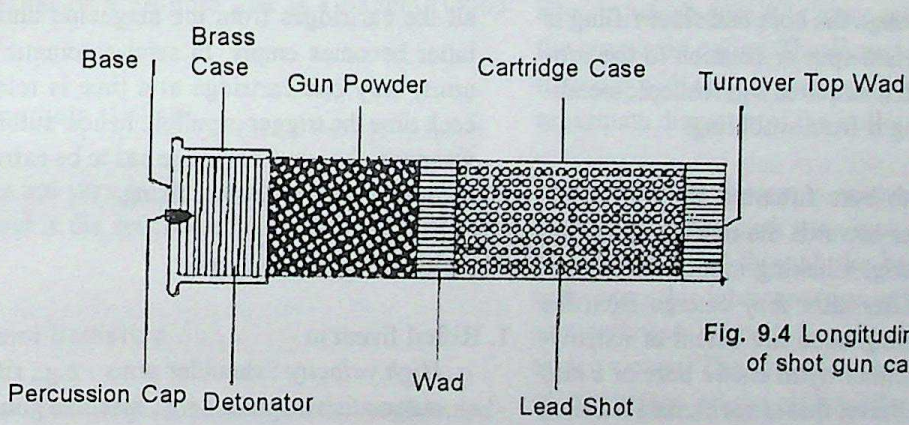

Fig. 9.4 Longitudinal section of shot gun cartridge

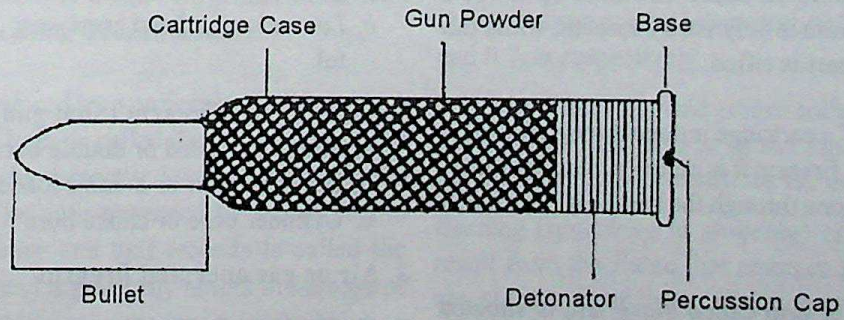

Fig. 9.5 Bullet of rifled firearm with case

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i) **Barrel**: It is a steel tube of varying length, and may be single or double. The *chamber* constitutes the rear end of the barrel, and lodges the cartridge. This part is called the *breech*. The front (distal) end of the barrel which is open to the exterior, is called the *muzzle*. The term *bore* refers to that part of the barrel through which the projectile makes its exit. The barrel may measure any thing from 2.5cm (1inch) to 90cms (3 ft). The tapering portion connecting the chamber with the bore is called the *leed* (*taper*). In rifled firearms, the interior of the barrel is scored by a number of parallel, spirally twisted grooves called *rifling*, whereas in smooth bore weapons the interior surface is smooth. The rifling may be either clockwise or anti-clockwise. A cross section of a rifled bore would reveal a series of elevations alternating with depressions (corresponding to the grooves). The former are called *lands*. The bullet is squeezed between the lands as it travels through the bore and since rifling is spiral, it imparts a spin or rotation to the projectile by which it acquires a gyroscopic steadiness, preventing it from wobbling.

In some smooth bore firearms, the barrel becomes narrower towards the muzzle end. This is called *choking*. Choking minimises dispersion of the pellets after they emerge from the barrel. Depending upon the extent of narrowing, it may be either a *full choke* bore or a *half choke* bore. A barrel that is not choked is called *cylinder bore*. In some firearms (**paradox guns**), the bore is only partly smooth, while the remaining part is rifled.

If loading of a cartridge is done through the muzzle end of a firearm it is called a *muzzle loader*, and if it is done through the breech it is a *breech loader*.

ii) **Bolt**: This is also called *block* and is situated towards the breech end of the barrel. As described earlier it consists essentially of a trigger, which when pulled releases the spring to activate the firing pin, which quickly moves forward to strike the base of the cartridge inside the chamber. The trigger is usually surrounded by a trigger guard to prevent accidental firing. In addition, there is a safety catch which prevents the gun from discharging even when the trigger is accidentally pulled.

The block also contains an *extractor* to remove the spent cartridge, and is fashioned into a grip or butt around the trigger mechanism, for holding the firearm. The block can be opened to insert a fresh cartridge into the chamber manually or automatically. In automatic firearms, the cartridges are arranged one below the other to form a *magazine*, which is attached to the block. If the trigger is pressed and the pressure is sustained, it will cause the continuous release of all the cartridges from the magazine until the latter becomes empty. In semi-automatic firearms, only one cartridge at a time is released each time the trigger is pulled. In non-automatic weapons, the spent cartridge has to be extracted manually each time after firing.

Classification of firearms

1. **Rifled firearm -**
   a. *High velocity* : shoulder arms - e.g., rifle automatic weapons – e.g., machine gun, sten gun
   b. *Low velocity* : hand arms – e.g., revolver, pistol
2. **Smooth bore firearm (Shot gun) -**
   a. Single barrelled or double barrelled
   b. Breech loader or muzzle loader
   c. Cylinder bore or choke bore
3. **Air or gas operated firearm**
4. **Country made firearm**

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CALIBRE

The *calibre* of rifled firearms is usually expressed in terms of the inner diameter of the barrel, in fractions of an inch. Thus, we have the 0.303 and 0.22 rifle, meaning that the calibre is 0.303 of an inch and 0.22 of an inch respectively. The measurement is made between two diametrically opposite lands and not between grooves.

The calibre (or *gauge*) of a shotgun is expressed in terms of the number of balls of lead which can be made out of 1 pound (450gms) of the metal, so that each fits exactly into the bore of the weapon. If 12 balls of the same size, can be accommodated, the calibre is referred to as 12 bore. The other commonly available calibre of shotgun is 16 bore. The actual calibre (in terms of inner diameter of the barrel) of a 12-bore gun is 1.850cm (0.729 inches) while that of a 16-bore gun is 1.685cm (0.663 inches).

Intermediate Ballistics

This is not very important for a forensic pathologist, and is the specialised field of a ballistics expert.

Terminal Ballistics

Forensic pathologists must have a thorough knowledge about Terminal Ballistics, which deals with the injuries resulting from firearms.

The wound produced by a projectile (*bullet* or *shot*) as it enters the body is called the *entry wound*, and that by which the projectile leaves the body is the *exit wound*. The path traversed by the projectile between the entry and exit wounds is called the *track*. Exit wound is generally absent in the case of shotgun injury, except in the region of the head.

RIFLED FIREARM INJURY

Entry Wound

The distance between the muzzle end of the firearm and the target is called the *range*. This may be

- Contact range, if muzzle end is in contact with body,
- Close range, if the range is within the distance travelled by flame
- Near range, if it is within the distance travelled by unburnt or partially burnt gun powder, and
- Distant range, if it is beyond the range of flame, smoke and gun powder particles.

During the bullet's attempt at perforating the skin while entering, due to the spin, the edge of the entrance wound may be abraded in the form of a collar, which is termed the *abrasion collar* or *areola*. In some cases there is contusion instead of abrasion, in which case it is more appropriately called the *contusion collar*. The diameter of the entry hole together with the abrasion collar may give the approximate diameter of the bullet.

The barrel of a firearm is generally lubricated between use. When such a weapon is fired, the bullet as it is propelled through the barrel, would naturally carry this lubricant (grease) on it, which subsequently gets deposited on the skin around the entrance wound. The spin of the bullet causes wiping of its surface on the skin while entering. This is called the *grease* (or *dirt*) *collar*. The abrasion and grease collars normally measure only about 0.3cm and 0.7cm respectively.

When both abrasion and grease collars are present, the grease collar is seen as the inner zone, while the abrasion collar constitutes the outer zone.

Burning (*scorching* or *singeing*) of skin and hair result from the flame that emerges from the muzzle, at the time of firing. Clothing around the entry wound may also show evidence of burning.

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Tattooing (*peppering* or *stippling*) results from grains of gun powder being driven into the skin, each grain acting as a minute missile. Tattooing is seen on the skin as small, discrete, black specks which cannot be wiped off.[5] The extent of tattooing will depend on the calibre of the weapon, the type of powder used, and the range. Tattooing may be absent if firing has taken place through clothing.

Blackening or *smudging* results from a superficial deposit of smoke on the skin. In other words, it is only a carbonaceous deposit on the skin, and hence can be easily removed by wiping with a wet sponge.[6] The intensity of smudging will depend on the calibre of the weapon, the type of powder used, and the range. Thus, the greater the calibre of the weapon, the wider the area of blackening and vice versa. Similarly, if black powder is used, the blackening will be more clearly visible, than if smokeless powder had been employed. Smudging may be absent on skin if firing has taken place through clothing. The presence of blackening, especially when smokeless powder has been used, may not be clearly visible to the naked eye. In such cases, infra red or ultraviolet photography will help to visualise it.

Since carbon monoxide is evolved on explosion of gun powder, the presence of this in the blood of the injured tissues at the entrance wound imparts a cherry red colour to the tissues.

A *lead ring* or *metal ring* around the entry wound results from the deposition of very small quantities of lead or other metal in the form of a ring or collar, as the projectile enters the skin. The lead ring can be appreciated radiologically or by neutron activation analysis.

Firearm wounds are generally easily recognised as such. But sometimes wounds caused by red hot pokers or a burning pointed stick may simulate bullet entry. However in these wounds, there will be no evidence of tattooing or cherry red colour. Conversely, glancing injuries caused by rifled firearms may be confused with incised or lacerated wounds.

i) **Contact shot**: It also known as a *point blank* shot. A contact wound over a dense area such as the cranial vault is generally large and cruciate (cruciform, stellate, or star shaped), due to explosive effects of the gases liberated. The imprint of the muzzle of the weapon may be found stamped on the skin. Burning, fouling by smoke, and tattooing are slight or absent in the adjacent skin, since all the components of the explosion are driven into the wound. The tissues are often saturated with carbon monoxide and therefore cherry red in colour. Cranial contact wounds are generally seen on the forehead or temple.

   Contact wounds over thin bone, chest, or abdomen are usually circular in shape, and are surrounded by abrasion or contusion collar. The overall diameter of the hole plus the collar represents the approximate diameter of the bullet. Surrounding hair is singed. The muscles around the track of the bullet may be cherry red in colour due to saturation with carbon monoxide.

ii) **Close shot**: Flame travels approximately up to 7.5 cms in the case of revolver or pistol, and 15 cm in the case of rifle. The wound appears as a circular hole surrounded by scorching, singeing, and smudging. These may be absent if firing has occurred through clothing. Abrasion collar, grease collar, and tattooing may be present.

iii) **Near shot**: The entry wound is circular or oval in shape. Unburnt powder grains and small metallic particles travel approximately up to 60cms in the case of revolver and pistol, and 75 cm in the case of rifle. In practical situations, tattooing is seen up to a maximum distance of about

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90cm. Singeing of hair and scorching are absent. Smudging can occur up to a range of 30cm. Beyond this it is not seen. Grease collar and abrasion collar are present.

iv) **Distant shot** (**Fig 9.6a**): The entry wound is circular with inverted margins. Scorching, tattooing, and smudging are all absent. Grease collar and abrasion collar are present. Distant shot suggests a range beyond self-infliction. The range in any case of gunshot injury can be estimated accurately by test firing using the same gun and similar cartridge, at different ranges and comparing the effects with the wound present in the victim.

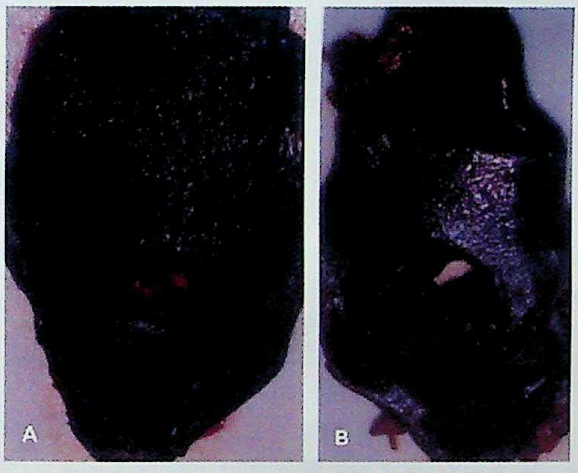

Fig. 9.6 Rifle firearm wounds a) Distant shot entry wound b) Exit wound

Exit Wound (**Fig 9.6b**)

Exit wounds vary greatly in size, shape, and configuration. They are usually larger than the corresponding wound of entry. Scorching, blackening, and tattooing are absent, as also abrasion and grease collars. Edges are invariably everted.

Salient differences between **entry** and **exit** wounds are mentioned in **Table 9.1**

Track

This is the path traversed by a projectile inside the body of a victim of gunshot injury, between the entry and exit wounds. In the case of low velocity weapons the track can be devious instead of straight. An X-ray prior to autopsy will assist significantly in locating bullets or pellets lodged in the body.[7] When a projectile traverses through dense tissues such as muscle, liver, or spleen, shock waves are generated causing tissue damage even at a considerable distance away from the track.

Table 9.1_Differences between Entry and Exit rifled firearm injury

Characteristic	Entry wound	Exit wound
Size	Smaller than the diameter of the bullet	Larger
Edges	Inverted	Everted
Abrasion and Grease Collar	Present	Absent
Burning, Blackening, Tattooing	May be present	Absent
Bleeding	Less	More
Fat extrusion	Absent	May be present
Cherry red colour	May be present	Absent
Lead ring	May be present	Absent

SMOOTH BORE FIREARM INJURY

Entry Wound (**Fig 9.7a & b**)

i) **Contact shot**: The contact shot wound in the case of shotguns is usually a large, irregular hole, resulting from the explosive blast effect. The edge of the defect is scorched by flame, and the skin surrounding it is blackened by smoke, and tattooed by unburnt powder. An imprint

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Fig. 9.7 Shot gun wounds a) Single wound (rat hole) b) Multiple wounds

abrasion produced by the muzzle end may be seen. The shot (comprising a bunch of pellets) passes into the body as a solid mass. The injured tissue is usually cherry red in colour. Shotgun injury of the cranium is large and irregular, and fissure fractures often radiate outward from the margins.

ii) **Close shot**: This produces a circular defect with irregular inverted borders. The edges are scorched due to flame, and smudged by smoke. A fairly wide zone of tattooing may surround the edges of the wound. Tissues often appear cherry red in colour. The pellets enter the track en-masse. Sometimes a shotgun may discharge parts of the cartridge case itself, such as fragmented cardboard, plastic or primer particles. At contact and close ranges, these may contribute to the wound.

iii) **Near shot**: The wound is circular or oval in shape. Smudging may be evident around the wound up to a maximum distance of 30 cm. Sometimes the wad produces mild abrasions if fired within a range of 30 cm. Tattooing is present over a wide area. The pellets travel in a compact mass up to a distance of about 45 cm, after which they begin to disperse. The entry wound is approximately 2.5 cm in diameter. If the shot enters at an angle less than 90 degrees, the shape of the wound may become triangular or semi-circular.

iv) **Distant shot** : Beyond a range of 2 metres, there will be no burning or blackening. Tattooing is also rare. But wad may be present in the wound (up to 5 metre range). The dispersion of pellets becomes significant at ranges over 2 metres. Thereafter, the spread increases progressively, and the central defect diminishes in size proportionately. There is an old rule of thumb which states that *the diameter of the spread in inches is roughly equal to the range in yards multiplied by 1.5*. At distant ranges (beyond 6-10 metres), the central hole may shrink to nothing. At such ranges the shot may not be lethal and the pellets, if they do penetrate the skin at all, may lie just in the subcutaneous tissue.

MEDICOLEGAL ASPECTS OF FIREARM INJURIES

Legally, firearms are classified as dangerous weapons, and mere possession without license or authority becomes a cognisable offence.

The following constitute some important medicolegal issues in firearm deaths :

- The kind of firearm used
- The range of firing
- The direction of fire
- The place from where firing took place
- The cause of death, and
- Whether the firing was an accident, homicide or suicide.

The nature of firearm

It is important to know whether the firearm used was a rifled weapon or a smooth bore weapon, and if it was a rifled weapon, whether it was a rifle, pistol, or revolver (**Table 9.2**).

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Table 9.2 Types of Firearms

1) **Pistol** is a miniature semi-automatic hand arm with a small barrel.
2) **Revolver** is a hand arm with a revolving cylinder filled with cartridges.
3) **Rifle** is a rifled shoulder arm for long range firing. Effective range may extend to a mile.
4) **Submachine gun** is an automatic rifled weapon, which also can be used as a self-loading firearm.
5) **Light machine gun** (LMG) is analogous to a rifle. It is of light weight and is capable of rapid uninterrupted fire with accuracy.
6) **Musket** is a smooth bore shoulder arm used for military purposes.
7) **Shot gun** is a smooth bore shoulder arm used for hunting.
8) **Air gun** and **Air rifle** are suitable for small game hunting. Lead slugs are used, the propelling force being obtained through compression of air.

An examination of the size of the bullet and its weight and calibre, the number, size, and direction of the rifling marks on it, and the kind of metal it is made of, will all give clues regarding the weapon used. For example, a bullet of 0.7 cm diameter generally indicates that it has been fired from a weapon with a calibre of 0.7 cm. The diameter of a bullet is measured with a micrometer.

Bullets may be recovered at the scene of a shooting incident, or from a dead body during autopsy. Although a detailed examination of the bullet recovered at autopsy is within the province only of a ballistics expert, the medical officer conducting the autopsy should observe the following points :

A bullet recovered from a dead body must not be washed or cleaned as this may remove the residue of any powder adhering to it. Instead, it should be dried without using heat and preserved for future examination by a ballistics expert. He must handle the bullet carefully. Forceps or other metallic instruments must not be used in handling a bullet, for it will cause not only artefacts such as scratches on it, but also obliterate the existing rifling marks. Rubber-tipped forceps are best for handling projectiles. The size of the bullet, its weight, calibre, the number, size and direction of rifling marks on it, the kind of metal of which it is made, any blunting of its nose, and other relevant details should be noted down. If the nose is blunted, it indicates that it had hit against a hard object. When a lead bullet strikes a person wearing clothes, the pattern of the weave of the clothing may sometimes be found stamped on the nose of the bullet. The bullet is marked for future identification, by inscribing one's initials on its base with a sharp pointed instrument, and not on its sides or nose, as this will obliterate other marks that may already be present. Bullets meant for future examination must be wrapped in absorbent cotton and preserved in empty cardboard boxes.

The suspect weapon and the crime bullet are both examined by a ballistics expert. To find out whether the bullet recovered had been fired from the suspect weapon, *test firing* is done. A test shot is fired from the weapon into a box called firing box, which is packed with cotton wool so that no distortion of the bullet occurs. The firing box is open at the side from which firing is done, and also at the opposite side. The bullet so fired (test bullet) is recovered. The test and crime bullets are then compared under a *comparison microscope*. This is a kind of microscope under which two objects can be compared simultaneously. If the rifling marks are identical on the test and crime bullets, the inference is that the suspect weapon was the weapon used for the commission of the offence. The number, width, and depth of rifling marks on a bullet can be accurately measured under a *mensuration microscope*.

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Examination of a spent cartridge case will also provide clues in identifying the crime weapon. In the case of a shotgun cartridge, the name of the manufacturing firm, and the calibre of the weapon for which it is intended, are generally imprinted on the casing. Empty cases of revolvers have a rim at their base, while those of pistols do not. The size of the spent case provides a clue regarding the calibre of the weapon. In a revolver, the empty case usually remains within the weapon, so that it is rare to find it at the scene of crime. If an empty case is seen at the scene of crime, as in the case of a pistol or a rifle, it is generally found a few feet to the right of the spot where the weapon was discharged. The firing pin will make a dent on the base, which is peculiar to that weapon. The breech may also leave its marks on it. The state of chemicals inside can assist in knowing whether it was recently fired or not. All these points can help to identify a crime weapon. For future identification, a spent case is marked by scratching one's initials on its side.

In a shotgun wound, the nature of the shot, e.g., lead pellets, glass beads, or stones, may help in identifying the weapon. Factory-made shotgun cartridges use lead shot, while home-made ones employ glass beads or stones. Lead pellets (and other kinds of shot), should be wrapped in absorbent cotton, placed in an empty cardboard box and preserved for examination.

Presence of *wad* at the scene of crime or in the dead body conclusively indicates that a shotgun had been employed. The diameter of the wad may give a clue regarding the calibre of the weapon. Wads are preserved in an empty card board box for future examination.

Stains on the body of a victim can also provide help. Rifled weapons employ cartridges with smokeless powder which does not cause much blackening of the skin, because it burns up entirely, while the black powder of shotguns causes extensive smudging.

The range of firing

The range of fire is deduced from the appearance of the entrance wound (*vide supra*). However, an accurate range of firing is usually better made out by test firing, than medical examination.

The direction of fire

If there is no deflection of the projectile, a line joining the entrance wound with the exit wound in a dead body, or the area of lodgement of the bullet will give the direction of fire. Cases of devious tracks, or deflection of projectile due to hitting an intervening object (*ricochet bullet*), can cause confusion in interpretation. An entry wound is circular if the projectile enters perpendicular to body surface and oval if entry is oblique. In the latter case, the edge on the side of the entry of the bullet appears bevelled, while the opposite edge becomes overhanging. In cases of oblique entry, the abrasion collar becomes eccentric, being wider at the side of entry.

The site of firing

This will depend upon the calculation of range and direction of firing.

Cause of death

Firearm injury can result in death due to haemorrhage, or damage to any of the vital organs such as the heart or the brain.

Manner of death

It must be deduced as to whether a particular case of firearm death was *accidental*, *suicidal* or *homicidal*. The important clues which can help to resolve this are mentioned in **Table 9.3**

Some other esoteric ballistic information is mentioned in **Box 9.1**

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Table 9.3 Differences between Accidental, Suicidal, and Homicidal firearm wounds

Feature	Accidental	Suicidal	Homicidal
Site of entry wound	Any part	Head or chest	Any part
Range	Close	Contact or close	Any range
Direction	Any direction	Upward or backward	Usually upward
Number of wounds	One	Usually one	One or multiple
Firearm residue on hand	Present	Present	Absent
Weapon at the scene	Present	Present	Usually absent
Location	Anywhere (usually home)	Usually home	Anywhere
Sex	Usually males	Usually males	Either sex
Motive	Absent	Depression, mental illness	Robbery, revenge, etc.

Box 9.1 Ballistic Trivia

Types of Bullet
- *Dum-dum bullet* – A type of bullet which is designed to explode on impact.
- *Incendiary bullet* – A hollow-point bullet which contains phosphorus.
- *Frangible bullet* – A bullet that is made of iron instead of lead, and fragments on impact.

Types of Lead Shot
- *Dust shot* – Consists of extremely fine particles.
- *Bird shot* – Consists of lead pellets of average diameter 3.5 mm. Used for small game hunting.
- *Buck shot* – Consists of large pellets of average diameter 6 to 8 mm. Used for big game hunting.

Unusual Ballistic Effects
- *Billiard ball effect* – Extensive dispersion of lead shot because of the pellets striking against each other in flight. May give erroneous impression about the range of fire.
- *Balling of shot* – The lead shot is bunched together for some distance during flight, resulting in much less dispersion than expected for that range.
- *Tandem bullet* – Sometimes, when a firearm is discharged, the bullet does not emerge. On pulling the trigger again, two bullets may emerge one behind the other. This happens in faulty or disused firearms.
- *Tandem (Duplex) cartridge* – A single cartridge contains two bullets one behind the other, so that a single shot produces two separate entry wounds. Used in military rifles.
- *Shored exit wound* – if the skin at the exit wound is supported against a wall, the wound may appear nearly circular with a surrounding margin of abrasion, simulating an entry wound.
- *Yawing bullet* – A bullet that has an unsteady trajectory during flight.
- *Tumbling bullet* – A bullet that tumbles end-on-end (somersaults) during flight.
- *Souvenir bullet* – A bullet that remains in the body of a surviving victim of gunshot injury for a long time without producing any deleterious effects.
- *Kennedy phenomenon* – Surgical intervention and alteration of a firearm wound, causing problems in medicolegal interpretation.

Methods of finding out whether a person recently discharged a firearm:
- Harrison and Gilroy's test.
- Neutron activation analysis.
- Atomic absorption spectroscopy.

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CHAPTER - 10

Regional Injuries

Easy it is to assess and quantify bodily damage, but who can understand the gravity of mental agony, or empathise with the unfortunate victim?

- **Justice Burton**, The Twists and Turns of Law

Mechanical injuries can assume varying significance depending on the location, and the structure or organ that is involved. It would be pertinent therefore to discuss some common injuries in a region-wise manner.

HEAD

Mechanical force applied on the head can injure the scalp, skull, meninges, brain, or related vessels, separately or in combination.

SCALP

Wounds of scalp can take the form of abrasions, incised wounds, lacerated wounds, contusions, or puncture wounds. They are rendered more visible if the scalp hair is shaven off. Abrasions are relatively uncommon over the scalp because of the presence of hair. Incised wounds produced by light cutting weapons, and chop wounds produced by heavy cutting weapons can be differentiated by the presence or absence of contused edges. Incised wounds of head bleed profusely. However, wound healing is rapid when compared to other regions. Due to the close proximity to the skull bones, scalp lacerations are often incised looking lacerations, which can be differentiated from true incised wounds only by using a hand lens. Crushing of hair bulbs and contusion of the edges help in confirming lacerated wounds. Like incised wounds, lacerations of scalp bleed profusely. Contusion of scalp can percolate and settle around the orbit, and be visible as a **black eye**, the so called *ectopic contusion* (**Chapter 8, Fig.8.6**). Superficial contusions appear as localised swellings on palpation, due to limitation by the fibrofatty tissue. Contusions deep to galea aponeurotica tend to become diffuse in nature due to the loose sub-aponeurotic tissue. Puncture wounds of scalp may be incised or lacerated puncture wounds, of which the latter are more common. Such wounds invariably enter into the skull cavity to become penetrating wounds.

SKULL

Fractures of skull could be due to direct impact (such as a blow), or due to indirect impact (such as a fall on the feet or buttocks). If the force of impact is less than the elasticity of the skull, in-bending of the bone at the site of impact alone is the result.[1] The bone subsequently resumes its original position. A fracture results when the impact is greater than the elasticity of the bone. Experiments have proved that a force of 5 foot pounds can be sufficient to fracture the skull. However, the occurrence of fracture also depends upon the relative thickness and strength of the bone in different regions.

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Classification

- Fissured fracture
- Comminuted fracture
- Gutter fracture
- Pond fracture
- Diastatic fracture
- Perforating fracture
- Cut fracture

Fissured Fracture (Fig 10.1)

It is a linear fracture involving both tables of skull, and is the commonest type of skull fracture.[2] Such fractures can be straight, curved or irregular. They can occur at the site of impact or some distance away, depending upon the temporary deformation of skull due to the applied force. They usually occur along the line of maximum stretching (Fig 10.2). Fissured fractures can also occur as radiations from the sites of other types of skull fracture. They are produced ordinarily when the head hits against a hard surface, or when an object with a large striking surface hits the head with sufficient force. A circular fissured fracture occurring around the foramen magnum is sometimes referred to as a **ring fracture**. Such a fracture results indirectly from the transmission of force through the vertebral column, as in the case of a fall on the feet or buttocks, or through the mandible as in the case of a blow to the chin. Ring fractures occur only in about 0.4 percent of skull fractures reported.[3] A fissured fracture which involves only one table of the skull is referred to as a **crack fracture**. It is usually the inner table which fractures in such cases, since it is more brittle.

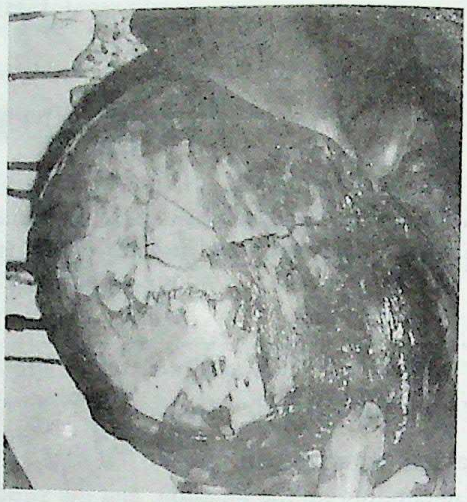

Fig. 10.1A

Fig. 10.1B

Fig: 10.1 Fissured fractures – A. Calvarium, B. Base of Skull

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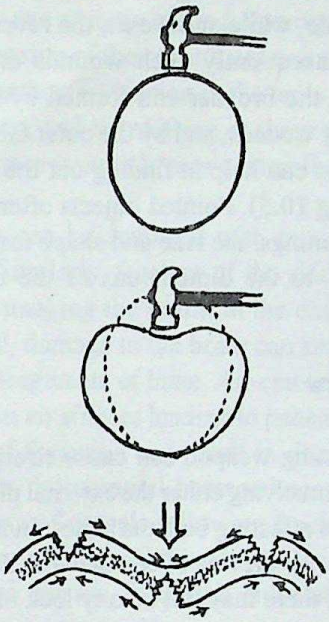

Fig: 10.2 Mechanism of fracture of skull

Comminuted Fracture

The term "comminution" refers to fracture division of a bone into several fragments. This results when a considerable force is applied over a relatively small area. Weapons with small striking surface like hammer are often the causative objects. Comminution can also be a complication of fissured fractures. Comminuted fractures can be with or without depression of the affected area. The former is called **depressed** comminuted fracture, while the latter is referred to as **non-depressed** comminuted fracture or merely as a comminuted fracture.

Depressed comminuted fracture (**Fig.10.3**) : Local impact causes the outer table to be driven inwards. If the force is not absorbed in the diploe, the inner table also gets invaginated into the cranial cavity - both plates therefore get comminuted, and thus a depressed comminuted fracture is produced. Such a fracture may provide a clue about the striking surface, i.e., the weapon used (**signature fracture**). For instance, a circular area of depressed comminuted fracture can indicate the application of a hammer.

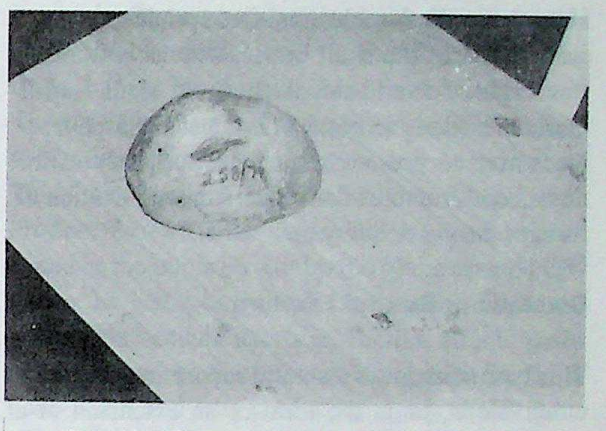

Fig. 10.3 Depressed comminuted fracture

Non-depressed comminuted fracture : In this type, there is no invagination of fractured segments. The fractured site appears in the form of several fissures arranged like a cobweb or "mosaic" (**Fig.10.4**).

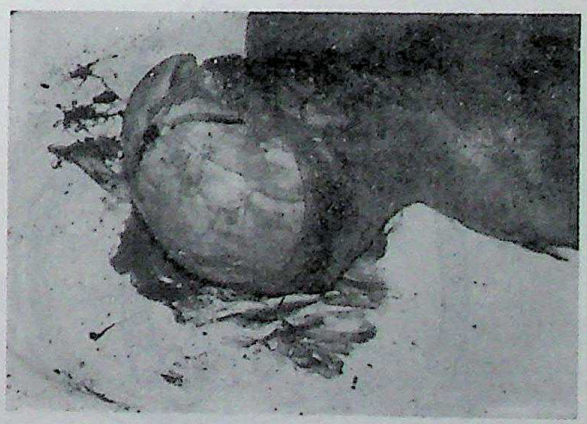

Fig. 10.4 Non depressed comminuted fracture

Gutter Fracture

If a weapon strikes the skull tangentially, the outer

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table alone may be fractured causing a deformity in the form of a gutter. The commonest example is a glancing bullet injury.

Pond or Indented Fracture[4]

Here the applied mechanical force produces a mere indentation of the skull bones, without comminution. This is due to the elasticity of skull, and is especially likely to occur in infants. Fissured fractures may be present around the periphery of the dent. Pond fractures can result from application of forceps during delivery.

Diastatic or Sutural Fracture

This type constitutes a sutural separation occurring either alone, or in the form of an extension of a fissured fracture.

Perforating Fracture

It results from penetration of the skull by a sharp pointed object or a bullet. In the latter case, the entry causes more damage to the inner table than the outer table, while at the exit, the reverse effect is seen. Consequently such wounds are funnel shaped with the broader end formed by the inner table in entry wounds, and by the outer table in exit wounds. This can help in finding out the direction of firing (**Fig 10.5**). Pointed objects often produce clear cut openings, the size and shape usually corresponding to the dimensions of the causative weapon.

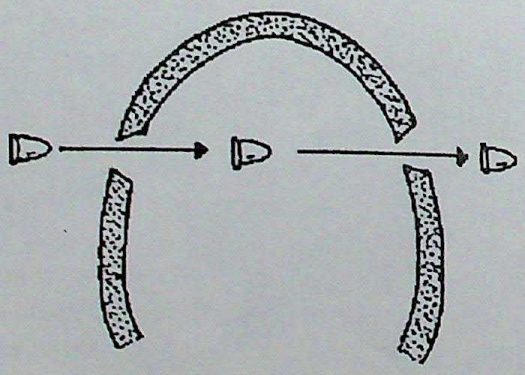

Fig. 10.5 Entry and Exit of a bullet through skull

Cut Fracture

A heavy cutting weapon can cause straight chops of the skull, involving either the external plate alone, or sometimes affecting both the plates. In these fractures, except for the line of contact with the edge of the weapon, there may not be any lack of continuity of the bony substance anywhere.

MENINGES

Any of the three meninges can be torn by edges of fragmented pieces of skull or by penetrating objects. However, pia and arachnoid can also be ruptured due to accumulation of blood from beneath.

BRAIN

The mechanism of damage to brain is explained well by **Holbourn's hypothesis**. In the case of a blow to the head, brain tissue gets injured when its constituent particles are pulled apart and do not join up subsequently. A shear strain type of deformation is thus produced in the plains of brain tissue. The forces transmitted from the skull to the brain act chiefly upon the surface of the brain. These forces are not evenly applied to the brain surface, but are maximally developed over those regions which are in close apposition to the projections of bony ridges or to the dural septa. Thus these regions of the brain usually suffer more damage than other areas.

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The actual physical disruption of cerebral tissue is caused by one or more of the following processes[5]: i) Compression of the constituent units while being forced together; ii) tension in the units while being pulled apart, and iii) shifting or shear strain which causes adjacent units to move laterally.

The brain can be damaged with or without skull fracture. Similarly, fracture of the skull can occur without damaging the brain. In the case of a fractured skull, damage to the brain can sometimes result from fragments of bone. Air can enter the ventricles from air sinuses leading to pneumocephalus. Fracture of foramina can result in damage to cranial nerves. Intracranial haemorrhages can occur from tearing of vessels following fractures, which can further damage the brain by increasing the intracranial tension. Bacterial entry can result in meningitis and brain abscesses.

It is said that in general, injuries to the brain in the back of the head are more likely to be fatal than injuries to the brain in the front part of the skull.[6]

Coup and Contrecoup Injury (Fig 10.6)

A coup injury is one which occurs at the site of impact, while contrecoup injury occurs at a site opposite to the point of impact. For instance, a blow over the occiput may bruise the frontal poles of the brain. An impact on a stationary head causes the skull to move immediately in the direction of the force, but the brain lags behind for a brief period. The skull hits the brain at the site of impact leading to the development of a coup injury on the brain. This type of coup injury is referred to as an *acceleration injury.* At the same time, vacuum is produced at the opposite site by the space created between the static brain and the forward displaced skull. This vacuum exerts a "suction effect" pulling the brain into that space to hit against the skull bone there, and thus producing injury to that part of brain leading to contrecoup injury formation.

Both coup and contrecoup injuries of brain can be in the form of contusions or lacerations.

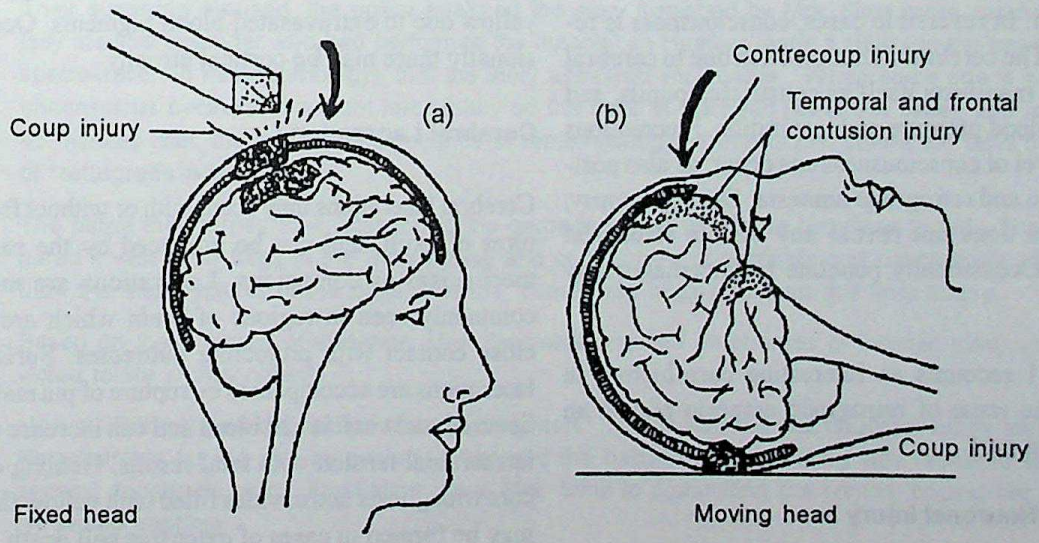

Fig. 10.6 Coup and contre coup injury

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When the moving head strikes against a stationary object, the skull is suddenly brought to a stop by the impact, but the brain continues to move forward for a brief period, thereby hitting against the skull. This type of coup injury is termed a *deceleration injury*. Forces leading to the rotation of skull causes the brain to slide or glide against fixed structures within the skull, resulting in shear strain injuries. Severe contrecoup injuries can result from a fall, even when coup injuries are absent or minimal. On the other hand, when the head is struck with an object, coup injuries are more common than contrecoup injuries. In a blow to a fixed head, contrecoup injuries may be totally absent.

Traumatic brain damage may take the form of **cerebral concussion, diffuse neuronal or axonal injury, cerebral contusion**, or **cerebral laceration**.

Cerebral Concussion (Commotio Cerebri)

This is a reversible or irreversible derangement of neuronal activity without demonstrable structural changes. In severe cases there can be total derangement of physiological activities leading to immediate death. In reversible cases, consciousness is regained. The cerebral irritation often due to cerebral oedema, manifests itself as constricted pupils, and altered blood pressure and respiration. Fluctuations in the level of consciousness can occur, as also post-traumatic and retrograde amnesia. During autopsy, the brain does not reveal any visible structural change. Occasionally, punctate haemorrhages may be seen.

Box 10.1 recounts an interesting case history in which the issue of retrograde amnesia played an important part.

Diffuse Neuronal Injury

Neurons and nerve fibres sustain injury in a diffuse manner when the force of impact is transmitted through the brain. This may be wholly or partly reversible. Rotational movements due to shear strains are the probable cause for this.

Diffuse Axonal Injury

Axons and vessels are disrupted as a result of impact. Deforming effect of shear strains is the main causative factor.

Cerebral Contusion

Cerebral contusions occur as circumscribed areas of brain tissue destruction, resulting from extravasation of blood in traumatised tissues. They are often multiple and found mostly in the cortex, but may also occur in deeper tissues. Cortical contusions are covered by a zone of intact cerebral tissue and are frequently wedge shaped. They are produced by distortion of skull or by rotational movements of brain in relation to skull. Absorbed shock waves of trauma and penetrating injuries can also produce contusions. Subsequent infection can convert a contusion to a brain abscess. Older contusions appear yellow due to extravasated blood pigments. Occasionally there may be cortical atrophy.

Cerebral Laceration

Cerebral lacerations may occur with or without fractures of skull, and can be produced by the same mechanism as contusions. Lacerations are most commonly seen in regions of brain which are in close contact with projecting buttresses. Surface lacerations are accompanied by rupture of pia mater. Severed pial vessels can bleed and can increase the intracranial tension with fatal results. Healing occurs with gliosis and cavities filled with yellow fluid may be formed in cases of extensive cell death.

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Box 10.1 The Sims Case (USA)

On the night of 29 April 1989, Robert Sims, a worker in a newspaper factory returned home to find his wife lying apparently unconscious on the kitchen floor. He revived her, and upon enquiry his wife recounted the following story – When she had gone out into the backyard earlier that night to empty out the garbage, a masked man suddenly grabbed her and forced her back into the house. He then hit her on the back with his hand (*karate chop*), as a result of which she fell unconscious and remained so for 45 minutes until her husband returned home and revived her.

To their consternation, when they went through the house they found their daughter Heather missing from her crib. The police were informed. After they arrived, they made a search of the house and discovered that nothing was stolen, and there was no disturbance at the scene. They waited for a ransom call which never came.

A few days later, a fisherman found a body wrapped in a garbage bag in a trash can several miles away. It was identified as that of Heather, on the basis of sole prints (foot prints) which were compared with those in the records at the hospital where Heather had been delivered. The body was autopsied by the local Medical Examiner who happened to be a lady who had specialized in neuropathology. She found evidence of smothering – bruising of inner aspects of lips and asphyxial signs.

The police began investigating the background of Robert Sims and his wife, and discovered that a few years earlier they had made a similar complaint relating to the disappearance of their younger daughter Laurie, while they were residing in another town. The skeletal remains of the baby in that case had been found, but the cause of death could not be ascertained, and the case remained unsolved.

Their suspicion aroused, the police analyzed the story furnished by Mrs. Sims more carefully. The lady medical examiner who had performed the autopsy on Heather made it clear (on the basis of her specialization in neuropathology), that the story appeared implausible. When some one is knocked unconscious because of a blunt force injury on the base of the head (as in this case), and recovers 45 minutes later, there is very little chance of remembering the events just before the blow because of "retrograde amnesia".

The police then turned their attention to the garbage bag in which the baby was found. They compared it with other bags in the Sims house and could prove scientifically and conclusively with help from the manufacturer of the bags, that the "crime" bag originated from that very house.

Based on these pieces of evidence, Mrs. Sims was arrested on charges of murder, tried, and convicted to life imprisonment.

Motive: Robert Sims did not like to have daughters. He ignored the first daughter totally and made life miserable for his wife by secluding her and the baby. The same thing was repeated with the second daughter's birth. Frustration drove Mrs. Sims to committing the crimes, hoping her marital life would improve.

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BLOOD VESSELS

Injury to intracranial blood vessels may manifest as any one of the following types of intracranial haemorrhages : **epidural (extradural), subdural, subarachnoid** or **intracerebral haemorrhage.**

Epidural (Extradural) Haemorrhage (Fig.10.7)

This type of haemorrhage occurs between the skull and the dura mater due to the rupture of diploic veins, venous sinuses or meningeal arteries. In the case of ruptured artery, the vessel most commonly involved is the middle meningeal artery, which usually gets cut at the sylvian point. A large haemorrhage, (2 cm or more in thickness), in the extradural space leads to rapid compression of the brain, leading to fatal involvement of the vital centres in the medulla. The initial injury to the brain may cause unconsciousness due to cerebral concussion. Recovery may follow, but later the patient becomes unconscious due to compression of brain from accumulated blood. The intermediary period of consciousness between the two periods of unconsciousness is termed as **lucid interval**. It is seen in approximately 27% of the cases. Extradural haemorrhage is almost always traumatic, and is more common in adults than in children.

Fig. 10.7 Extradural haematoma

Subdural Haemorrhage (Fig.10.8)

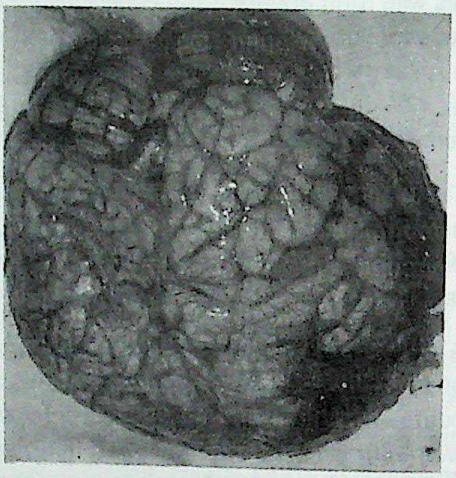

Fig. 10.8 Subdural haematoma

This type of haemorrhage occurs between the arachnoid and dura mater. This could arise from tears in the dural venous sinuses or cortical veins, but the commonest cause is rupture of bridging or communicating veins. It can occur in either closed or open head injury. Rapid accumulation of blood causing immediate brain compression is termed as *acute subdural haemorrhage*. In *subacute subdural haemorrhage*, the symptoms appear 2-14 days after injury. *Chronic subdural haemorrhage* takes several weeks for symptoms to commence, and the accumulation of blood is minimal to start with. Subsequent increase in the volume of the extravasation is characteristically slow. Subdural haemorrhage from natural causes is often bilateral, while the traumatic variety is usually unilateral. Subdural haematomas invariably become organised and encapsulated, into which bleeding can occur periodically from rupture of newly formed capillaries. Repeated bleeding and organisation causes progressive increase in size, and gives rise to a laminated appearance.

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Unconsciousness, ipsilateral pupillary disturbance, hemianopia, speech defects, contralateral hemiparesis, and urinary disturbance are the common features of acute subdural haemorrhage. Lucid interval is occasionally seen.

Subarachnoid Haemorrhage (Fig 10.9)

This type of haemorrhage occurs between the arachnoid and the pia mater, and usually arises from the rupture of cerebral arteries or cerebral veins. Blows to the neck can cause lacerations of vertebral artery with dissection of blood superiorly into subarachnoid space.[7] The blood mixes with the CSF and gets distributed over the whole of the brain surface. Subarachnoid haematoma formation is extremely rare. Spontaneous subarachnoid haemorrhage can occur from natural causes as in rupture of congenital (berry) or infective (mycotic) aneurysms, in arteriovenous malformations, and also in bleeding disorders. Even minor trauma can precipitate subarachnoid haemorrhage if the arteries are weak, as in atherosclerosis. Minor trauma can also rupture basilar arteries in chronic alcoholics.[8] Subarachnoid haemorrhage is said to be the most common intracranial lesion following injury.

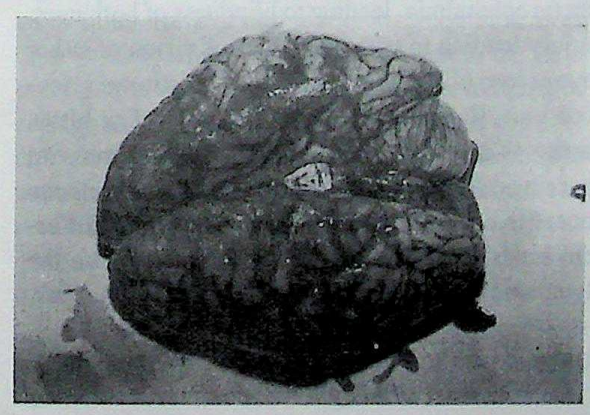

Fig. 10.9 Subarachnoid haemorrhage

It can be unilateral or bilateral, and may be localised or diffuse. Blood in the subarachnoid space is usually removed by phagocytosis, following the lysis of blood cells. About one month after injury, only yellowish brown areas of discolouration of the arachnoid may be left behind, which are referred to as **plaques jaunes**.

Intracerebral Haemorrhage (Fig 10.10)

This may be either a **parenchymatous haemorrhage**, or an **intraventricular haemorrhage**. Haemorrhage in the brain tissue (parenchymatous haemorrhage) arising directly from the trauma usually occurs near the surface. It may be punctate, echymotic or larger (haematoma). A single deep seated parenchymatous haemorrhage is usually due to a disease process, and is often located in the internal capsule, basal ganglia, or pons. Multiple punctate haemorrhages may be seen in bleeding disorders, leukaemias, septicaemia, typhus, Caisson's disease, fat embolism and Wernicke's encephalopathy. Traumatic intracerebral haemorrhages may or may not be associated with skull fracture. Those haemorrhages occurring along the axis of transmission of mechanical force from coup site to the contrecoup site are termed *intermediate coup lesions*. Occasionally, it takes several days for the haemorrhage to develop after a traumatic episode. Sudden death is uncommon. Minute haemorrhages occuring in the brain of a boxer due to repeated punches on the head can lead to mental changes and unsteady gait. This is called **punch drunkenness**.

Intraventricular haemorrhages are usually extensions of parenchymatous haemorrhages. However, they can occur as isolated events as in severe trauma, or from vascular malformations of choroid plexus.

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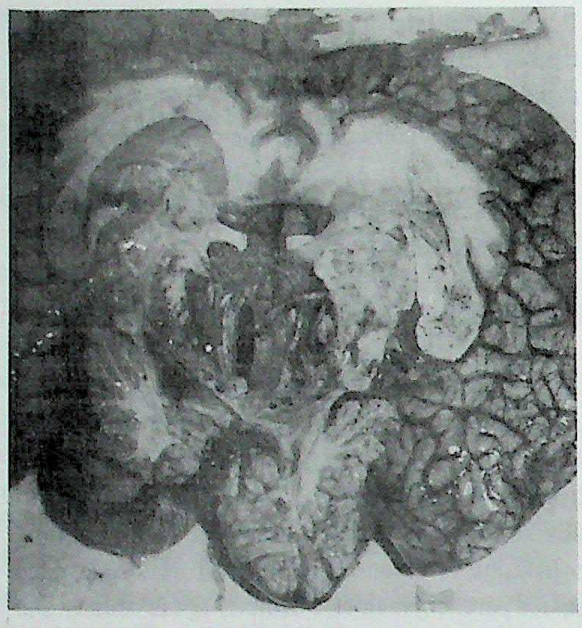

Fig. 10.10 Intracerebral haemorrhage

FACE

Injuries of face which cause permanent disfiguration represent *grievous hurt*. Such permanent disfiguration may be due to scar or keloid formation, or due to derangement or loss of tissues. Pulping of face can result from vehicular run over injury, or blunt impact by a heavy stone.

MOUTH AND NOSE

Abrasions and contusions in or around the mouth and nose could suggest forceful opening of the mouth to administer something, or forcible closure of mouth and nose as in smothering. Superficial lacerations of inner aspects of lips can occur due to forceful apposition of lips against the teeth, especially against the free edges of the latter. Injuries to lips can also result from blunt impact such as fisting. Occasionally, the lips or nose may be cut off or bitten off as a revengeful act.

Blunt trauma as in the case of a fall or blow, can cause injuries to nasal bones and teeth. Injuries which lead to fracture of jaw bones or nasal bones, or fracture/dislocation of teeth constitute grievous hurt, and are often accompanied by contusions or lacerations of lips and gums. If the cribriform plate of ethmoid bone is fractured, anosmia may result. Partial detachment of nasal mucous membrane leading to nasal bleeding occasionally occurs after blunt trauma to head, even without direct involvement of nose.

EYES AND EARS

The term **black eye** refers to accumulation of blood around the eyeball and eye lids which manifests as a darkish discolouration around the eye. This can occur directly from blunt trauma over the eye, or indirectly as an *ectopic contusion* especially from the scalp. Trauma can also cause injuries to cornea, iris, lens, and retina, or haemorrhage into anterior chamber. Retinal detachment and traumatic cataract are not uncommon sequelae of trauma. Penetrating injuries occasionally involve the eyes, sometimes leading to evisceration. Infections of eyeballs can spread to the brain and can lead to death by cavernous sinus thrombosis or meningitis. The uninvolved eye may suffer from sympathetic ophthalmia, leading to blindness of both eyes. Injury leading to permanent loss of vision of either eye or loss of hearing of either ear constitutes grievous hurt. Ears may be cut or chopped off or bitten away as an act of revenge, or to forcibly remove ear rings. Abrasions, contusions and lacerations can occur to one or both ears, from accidents of different kinds. A hard blow to the ear can rupture the tympanic membrane and cause deafness.

CHEEK

Abrasions, contusions, and lacerations of cheek can occur from blunt trauma. Bite marks can result from an assault, or may be the offshoot (ironically) of an

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act of love (*love bite*)! Cheek bones (malar bones) can fracture from direct or indirect violence. The fracture line in such cases is usually horizontal, and involves the posterior third of the malar skeleton.

JAWS

Fractures of mandible are commonly vertical, and invariably result from direct trauma. A blow to the point of the chin can indirectly cause a *ring fracture* of the base of the skull. Comminuted fracture of the maxilla is not an uncommon occurrence in blunt force injuries to the face. Occasionally there may be transmission of force to the cranial cavity leading to intracranial haemorrhages. Blood and dislocated teeth resulting from fracture of jaw bones may be ingested or even inhaled causing choking and death.

NECK

Superficial wounds of the neck may or may not cause serious bleeding, but penetrating wounds, incised wounds, and deep lacerations produce copious bleeding due to severance of carotid and jugular vessels. Direct impact to hypopharynx can fracture the thyroid cartilage. Hyoid bone can fracture from blunt impacting force, or from blunt constricting force, as in manual strangulation. Scratch abrasions in the form of nail marks are good indicators of throttling, while a pressure abrasion in the form of a ligature mark is pathognomonic for hanging or strangulation. A blow on the front of neck can precipitate vagal inhibition which can result in sudden unconsciousness or death. Choking due to blood can result from injuries to trachea and larynx. Laryngeal oedema is another serious complication of trauma to these areas.

Incised wounds of the throat are not uncommon. Such *cut-throat* wounds (**Fig 10.11**), can be suicidal, homicidal, or rarely accidental. In a suicidal case, the person usually holds the weapon in his right hand and starts the incision from the left side of neck drawing it to the right. Tailing of the wound is therefore seen on the right side. Thyroid membrane and muscles are usually cut and the wound slopes towards the floor of the mouth. Carotid arteries are not frequently injured as they slip backwards when the head is extended. Bleeding is usually venous, and loss of consciousness is gradual. However death may take place quickly from air embolism, due to air being sucked in by negative pressure in the veins. A person attempting suicide generally makes repeated horizontal, parallel, shallow, half-hearted cuts on the neck initially, before he picks up enough courage to make the final lethal cut sweeping across the neck. These preliminary shallow cuts which merge with the main wound represent hesitation or nervousness, and are therefore called **hesitation cuts, exploratory cuts, feeler strokes** or **tentative cuts**. A homicidal cut throat wound is invariably very deep, and obviously lacks hesitation cuts, though cases have been reported where superficial cuts resembling hesitation cuts have been present along with the main wound.[9] The direction is usually horizontal. The wound is often situated at the lower part of the neck and slopes away from the floor of the mouth. Carotid arteries are often severed.

Fig. 10.11 Cut-throat wounds

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Sometimes, air from wounded respiratory passages enters into the subcutaneous space resulting in *subcutaneous emphysema*, which may dissect down into the mediastinum leading to *mediastinal emphysema* and subsequent respiratory obstruction. The dome of the pleura if penetrated through the neck can cause pneumothorax. Aphonia and hoarseness of voice can occur if recurrent laryngeal nerve is involved, resulting in the cadaveric position of vocal cords. Injuries to postero-lateral aspect of neck can involve the brachial plexus leading to loss of function of the arm or hand. Infections from neck can spread to the mediastinum, which may cause a mediastinal abscess. Speech is impossible in cases of open wounds of larynx and trachea below the level of the vocal cords.

CHEST

Chest, although very elastic, becomes more fixed as age advances, and what would have been merely a temporary alteration in shape which would return to normal, will fracture under compression.[10] External and internal injuries of chest often coexist, though this need not be the case always. Thus serious internal injuries can occur without any external evidence of the same. Impact of a considerable nature can give rise to alternate compression and decompression. This leads to initial congestion followed by laceration and haemorrhage due to rupture of lung alveoli. Such sudden and swiftly acting trauma may not leave behind any external manifestation, but can still prove fatal after a lapse of time.[11] Injuries of the chest may be penetrating ("open") or non-penetrating ("closed").

CHEST WALL

Breasts are common sites for wounds originating from a sexual act, e.g., nail marks and love bites. As far as fractures of ribs are concerned, they usually occur in regions of maximum stretch. Thus anteroposterior compression of chest which causes maximum stretch at mid-axillary level produces fractures in the mid-axillary line, while side to side compression causes maximum stretch at the necks and costochondral junctions resulting in fractures at these sites. The 4th to 8th ribs are the commonest ribs to fracture. However, localised blunt trauma with reasonable force can involve any rib at any site. Fractured ends of ribs sometimes project outwards or inwards causing injuries to the overlying or underlying membranes, tissues, muscles, or organs. Lacerated wounds involving chest wall and parietal pleura can lead to "open" pneumothorax. Multiple rib fractures may give rise to **flail chest** or **stove-in chest** with consequent paradoxical respiration. Osteoporosis, osteomalacia and secondary malignancy are associated with fragility and easy fracture of ribs. 3rd to 5th ribs of left side are often involved in external cardiac massage. Such fractures usually occur at costochondral junctions and are associated with negligible amount of blood infiltration.

Sternum is a bone which is not easily fractured. But it can fracture from direct local force as in the case of an impact with the steering wheel of a vehicle in a collision accident. Such fractures usually occur at or near the manubrio-sternal junction. Fractures of sternum are usually transverse in nature. Forces which result in hyperflexion or hyperextension of sternum are likely to result in fracture. Severe blows on the chest wall may produce concussion of the chest, shock, and death, even in the absence of structural damage to the viscera.

LUNGS AND HEART

The lungs or the heart may get injured by blunt force applied on the chest, or from penetrating forces. They are especially susceptible to contusions or lacerations.

Lung injury can be gross or microscopic, focal or diffuse, and located centrally or peripherally, or

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both. Trauma of substantial degree can produce multiple small lacerations of lung, or a single large irregular cavity, the latter being termed "traumatic cavitation." The pleura may or may not remain intact. Injury to lungs can result in pneumothorax, haemothorax, haemopneumothorax, interstitial and subcutaneous emphysema, mediastinal emphysema, or obstruction of air passages. An injured lung may collapse.

Injury to heart can result in serious consequences. As far as bleeding is concerned, injuries of atria are more dangerous than ventricles, and those of right ventricle more dangerous than left ventricle. The right ventricle is more prone for injuries, since it forms the major part of anterior surface of heart. If the injury to the heart is obliquely placed, there could be flap formation as a result of which bleeding is minimised, since the flap acts as a valve of occlusion during contraction of heart. Penetrating injuries of heart inevitably penetrate the pericardium leading to accumulation of blood in the pericardial sac (haemopericardium), and subsequent escape from pericardial sac to pleural cavity (haemothorax). In blunt injuries, the pericardium usually remains intact, while the heart itself may rupture leading to development of haemopericardium. Rapid accumulation of as little as 250-350 cc of blood can cause **cardiac tamponade**, i.e., progressive limitation of the pumping action of heart and eventual cardiac arrest. Rupture of heart is more common in young individuals. Right atrium is most commonly involved in traumatic rupture, followed by right ventricle. In the old, blunt injuries can precipitate myocardial infarction. A diseased heart may rupture even with trivial trauma.

Blunt injuries can also cause contusion of heart walls, septae, and papillary muscles. They can give rise to ventricular fibrillation or progressive circulatory failure. In the case of a pericardial tear, the heart can herniate through it and get compressed - *strangulation of heart.*

Oesophagus, trachea, bronchi, bronchioles, aorta, vena cavae, and pulmonary arteries can be injured by blunt or penetrating trauma. Rupture of aorta is usually transverse, and may be complete or partial (involving only the intima). It frequently occurs at the junction of the arch with the descending part. Ruptures of oesophagus usually occur in the form of longitudinal splits. The most common site of rupture of diaphragm due to blunt trauma is at or around the central tendon of the left side. Immediate or delayed herniation of abdominal viscera into the chest can occur in such a case. Injury to thoracic duct can lead to chylothorax.

ABDOMEN AND PELVIS

ABDOMINAL WALL

The anterior abdominal wall easily yields to pressure, and hence usually escapes unscathed from blunt trauma. Contusion of anterior abdominal wall is therefore very rare, except in severe crushing injury as in the case of run over injury by a vehicle. However, abrasions occur quite commonly. The following are some important points worth remembering:

- Blunt trauma can result in extensive injuries of abdominal viscera even in the absence of any external injury.
- The actual total depth of a penetrating wound of the abdomen can be exaggerated due to the yielding nature of anterior abdominal wall.
- A blow to abdomen can cause death due to vagal inhibition.

VISCERA

Any of the abdominal or pelvic viscera can get injured due to blunt or penetrating trauma. In severe crushing force, there can be sudden shock, haemoperitoneum, or peritonitis.

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Oesophagus, Stomach and Intestines

Rupture of stomach usually occurs in the pyloric region along the lesser curvature. This is due to deficient muscular layer in this region, as well as due to lesser elasticity. Since stomach wall can be contused from blunt trauma, delayed rupture can occur in such contused areas. Spontaneous rupture of stomach is extremely rare. Dissolution of distal end of oesophagus (oesophagomalacia), gastric fundus (gastric malacia) or both, may take place in some persons hours or days after sustaining cerebral trauma. This is believed to be brought about by neurogenic stimuli arising from the parasympatholytic centre of diencephalon.

Intestines can rupture from blunt force either at the point of impact, or some distance away. The former shows clean edges, while the latter has a ragged appearance. Transverse ruptures of mesentery can lead to gangrene of a segment of intestine by transecting the supplying artery.

Liver and Spleen

Liver is an organ which is very vulnerable to injury. Rupture mainly occurs over anterior and inferior surfaces of the right lobe. The direction of rupture depends upon the direction of pressure. For example, if the force compresses the lower part of the liver, a transverse rupture results. If the right lobe alone is compressed, a vertical rupture occurs between right and left lobes. Hepatic ruptures can be transcapsular, subcapsular or central. Ribs can also cause injury to liver. Diseased liver (e.g., due to fatty change, malaria, etc), ruptures easily by trauma. Ruptured liver usually causes substantial haemoperitoneum.

Spleen is easily injured from trauma due to either blunt force or penetrating injury. The degree of damage varies from laceration of capsule to pulverisation. Bleeding is characteristically profuse. Malarial or leukaemic spleen is extremely susceptible to traumatic rupture.[12] Such a diseased spleen can sometimes rupture in situations of strong muscular contractions as in coughing or defaecation. The concave surface is more prone for rupture. Splenic ruptures result in haemoperitoneum. Ribs can cause laceration of spleen.

Urinary Tract

Rupture of urinary bladder often accompanies pelvic fractures, especially if the bladder happened to be full at the time of the incident. Even violent muscular contractions can lead to rupture of a full bladder. Ruptured bladder is usually accompanied by extraperitoneal extravasation of urine or blood. Shock, bleeding, peritonitis and urinary tract infection are common complications.

Urethra usually ruptures in the region of the membraneous part, and may accompany pelvic fracture, or result from the application of blunt force in the perineum. Female urethra may be ruptured during a violent act of rape. Falling astride a projecting object is a frequent reason for rupture of male urethra.

Lacerations of the kidneys can be transcapsular, subcapsular or transrenal.

Genitalia

External genitalia can suffer different types of injuries, especially related to violent sexual act or sexual jealousy. Squeezing the testicles can be fatal due to reflex cardiac inhibition. Criminal amputation of male genitalia can cause death from bleeding. Self-inflicted injuries to genitalia are sometimes encountered in insane persons. Attempted criminal abortion or self-introduction of foreign objects can lead to injuries of female genitalia.

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LIMBS

Any type of wound can occur to the limbs, from blunt injuries to sharp incisions. Direct and indirect trauma can produce different types of injuries to muscles. Tetanus, gas gangrene and renal failure may follow. Muscles may sustain severe crush injuries even though the skin may be intact (due to elasticity of the latter). Thighs with intact skin can accommodate even up to one litre of blood resulting from rupture of vessels due to a crushing force This can be fatal. Even multiple superficial soft tissue injury due to blows or kicks can cause death, though the bones may be intact.[13]

Ligaments can undergo avulsion. Blunt injury to the limbs is extremely common, especially in traffic accidents.[14] Fracture of femur or the bones of the lower leg resulting from the impact of a vehicle against a pedestrian is often referred to as a **bumper fracture**. Wounds sustained by a victim while trying to defend himself from an attack by raising his hands, or attempting to wrest the weapon away from the assailant, are called **defence wounds**. They are usually seen on the palms and inner aspects of forearms.

VERTEBRAL COLUMN

A useful categorization of vertebral column injuries is to divide them into fractures, dislocations, and fracture-dislocations.[15] Such injuries can occur to the vertebral column due to direct or indirect mechanical force, which may or may not involve the spinal cord. Commonest site of traumatic damage to the vertebral column is the lumbar region. Spinal cord is rarely injured without associated fractures of vertebral column. A sudden drop from a height of more than 5-7 feet in a case of hanging can cause dislocation between 2nd and 3rd, or 3rd and 4th cervical vertebrae, resulting in sudden death. Dislocation of atlas, with or without fracture of odontoid process, is a potentially lethal injury. Fractures of vertebrae can be one of the following three types : i) *Wedge compression fracture* (vertebral bodies are compressed from above and below) ii) *Comminuted fracture*, and iii) *Fracture-dislocation* (sliding of one vertebra over another). A direct impact usually causes comminuted fracture, while an indirect impact, as in a fall on the feet or buttocks causes wedge compression fracture. Hyperflexion or hyperextension of neck can result in cervical vertebral column injury and is called **whiplash injury**. Fatal contusion or laceration of the spinal cord can occur in whiplash injuries in the absence of any fracture of the vertebral column. Involvement of the cord above the level of the 5th cervical vertebra can be fatal due to damage to phrenic nerve. Lower level involvement can cause quadriplegia.

Paraplegia, quadriplegia, and respiratory and urinary infections invariably follow vertebral column and spinal cord injuries. Sacral segment involvement can lead to impotence in a male victim. Blunt force over vertebral column can lead to concussion of spinal cord without visible injury to vertebrae. This is called **railway spine** due to its frequent occurrence in railway accidents. Commonest region affected is the lumbo-sacral spine resulting in paraplegia, bladder disturbances, and impotence. Since concussion is spontaneously reversible, these abnormalities are usually only temporary.

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CHAPTER - 11

Thermal Injuries

"From the ashes of many a crime, riseth like a phoenix, the face of nemesis".

- Sir John Farquhar

Heat is a form of energy which when transferred to the body produces thermal injury in the form of **burns** or **scalds**, the former resulting from dry heat and the latter from moist heat. Thermal injuries therefore result from the application of flame, hot liquids, gases, solid heated substances like metal or glass, lightning, electricity, ultraviolet or infrared rays, X-ray and corrosive chemical substances.[1]

Scalds are produced due to application of moist heat to the body in the form of boiling liquid or steam. It is classified into first, second and third degree scalds. In the *first degree*, there is reddening of the skin, in the *second degree*, there is blister formation, and in the *third degree*, drying and desiccation of deeper tissues.

When external heat is applied to the human body, the extent of damage depends on the applied temperature, the time for which it is applied, and the ability of the body to conduct excess heat. Temperatures as low as 44°C can cause damage if there is prolonged period of contact.

BURNS

MECHANISM OF DEATH

Death due to burns may be the result of **neurogenic shock** due to pain, **hypovolemic shock** due to loss of fluid, **toxaemia** as a result of absorption of toxic material from the burnt site, or **septicemic shock** due to infection. Death occurring within 24 hours is usually due to neurogenic shock, hypovolemia, or toxaemia. Delayed deaths, which occur after 72 to 96 hours, are usually the result of infection, or renal damage, or haemorrhage from erosions (*Curling's ulcers*) of gastro-intestinal tract.

CLASSIFICATION

Based on depth -

Dupuytren classified burns into *six degrees* depending on the severity of the burns.

I° Burns are characterised by erythema (reddening of the skin), followed by desquammation of superficial layer of epidermis.
II° Burns are characterised by blister formation.
III° Burns result in destruction of the epidermis.
IV° Burns result in destruction of the whole thickness of skin.
V° Burns involve destruction of muscles.
VI° Burns result in the destruction of bone, nerve trunks, etc.

Hebra classified burns into *three degrees*, merging every two degrees of Dupuytren into one. **Wilson** put forth a simpler and clearer classification (*vide infra*). On naked eye examination, the depth of the burn is difficult to make out, and hence

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he considered Dupuytren's classification to be of limited value in the practical situation.

Wilson's Classification[2]

First Degree: *Epidermal burns*

In this type of burn injury, the lesion is confined to the epidermis. There may be blistering without loss of epidermis. There is capillary dilatation and transudation of fluid into the tissues, causing swelling. If the blister bursts, a reddened base is seen. First-degree burns heal without scarring.

Second Degree: *Dermoepidermal burns*

There is destruction of full thickness of the skin. The epidermis is coagulated or charred. There is a central zone of necrotic tissue surrounded by first-degree burns or hyperaemia. The central necrosis sloughs off and the epidermis grows in from the margins. Scarring is inevitable, and due to formation of contractures, there is disfiguration.

Third Degree: *Deep burns*

There is destruction of deeper tissues below the skin surface. The skin is totally destroyed, though in some deep burns, islands of intact dermis may be found. Damage to subcutaneous fat, and loss of muscle, bone, etc., is seen.

Based on surface area –

The Rule of Nine

For the assessment of percentage of burns (used when burns are superficial in nature). **Table 11.1** summarises the calculation of surface area involved, on the basis of the **rule of nine**, while **Fig 11.1** is a diagrammatic representation.

Table 11.1 Rule of Nine

Body Part Affected	Percentage of Burns
Head & Neck	9
Right upper extremity	9
Left upper extremity	9
Right lower extremity	18
Left lower extremity	18
Front of the trunk	18
Back of trunk	18
Genitals	1

It is important to remember that the calculation is slightly different in children less than 5 years old. Since the head is proportionately larger in a child relative to the rest of the body, as compared to that of an adult, the percentage allotted for the head and neck is *18%* and not *9%*. Since the lower limbs are proportionately smaller, the percentage allotment is *13.5%* for each lower limb, i.e., *27%* for both put together, instead of *36%*.

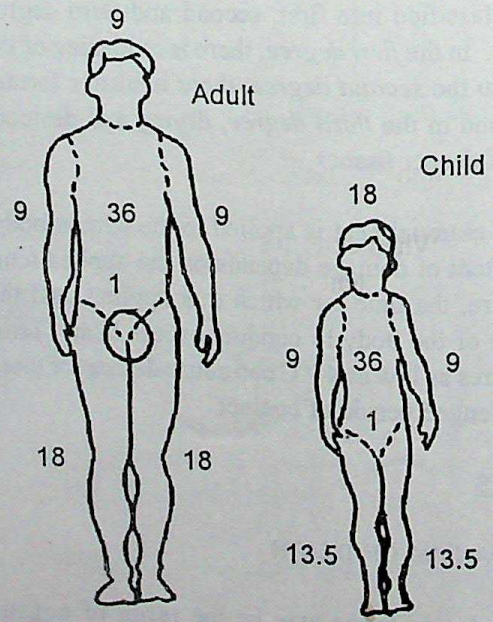

Fig 11.1 The Rule of Nine

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GENERAL FEATURES OF BURNS

A burn injury will be more severe if the heat applied is of great intensity. However if the body is exposed to heat for a long time, it will show severe burn even if the intensity is less. The severity of burn also depends on the area involved. Burns affecting the face, genitals, or lower part of the abdomen are more dangerous than burns in other parts of the body. Infants and elderly individuals are more susceptible to complications of burns.

Heat Haematoma

A haematoma may form in the extradural space between the skull and the dura mater when the cranium is exposed to tremendous heat. It resembles a traumatic extradural haematoma, and may originate from venous sinuses, or from emissary veins in the diploe of the skull.

It can be differentiated from traumatic extradural haemorrhage by the presence of charring of the skull. The clot itself is soft and friable with honeycomb appearance, and follows the distribution of charring of the outer table of the skull.

Heat Ruptures

Heated skin contracts and splits. Such defects may appear to the inexperienced observer as antemortem wounds. They are usually seen on the elbows, knees, and over the extensor surfaces of other joints, as well as on the head.

Heat Rigor

Heat rigor is observed mainly in the muscles. This phenomenon is seen particularly in cases with extensive burn injury. It is due to coagulation and denaturation of proteins. The posture of the body in such cases will demonstrate generalised flexion of the trunk, arms, and legs. The upper limbs are held out with the fingers curled inwards, akin to the characteristic attitude of a pugilist (boxer) who is ready for a bout. Hence, this is sometimes referred to as the **pugilistic (boxer's or fencer's) attitude.**

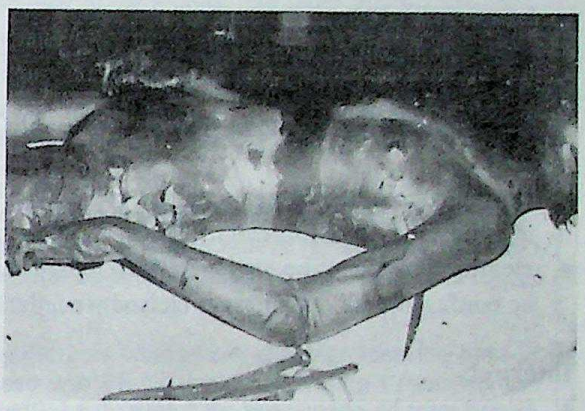

Fig 11.2 Extensive burns with carbonisation

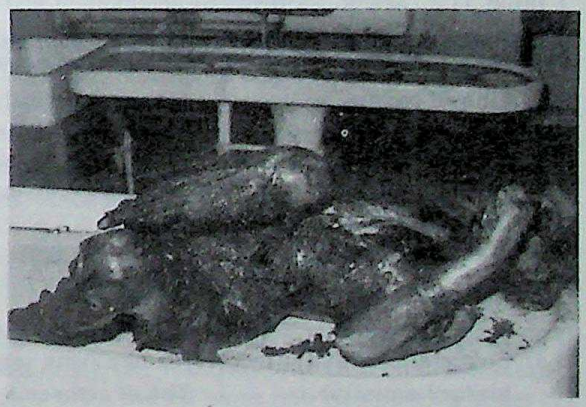

Fig 11.3 Pugilistic attitude

AUTOPSY FINDINGS

External -

- The body may demonstrate "pugilistic attitude" (which is however seen in both antemortem and

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postmortem burns). Table 11.2 outlines the salient differences between antemortem and postmortem burns.

- Antemortem burns appear reddened and blistered. Hair is singed or burnt. Even pubic hair, eyelashes, and eyebrows may be involved.
- In case of scalding, the body and clothes may be wet. If the scalding fluid has trickled down, the scald effect diminishes along its track, being maximum at the site of initial contact with skin.
- Burning of clothes, singeing of hair, charring of the body, etc., are not seen in scalds.
- Heat ruptures may be evident, which must not be confused with antemortem incised wounds.

Internal -

- Blood may be *cherry red* in colour, due to the presence of carboxyhaemoglobin.
- Extradural heat haematoma may be seen.
- The brain is congested, and may demonstrate a "cooked" appearance.
- Soot particles or carbonaceous material may be seen in the respiratory passages due to smoke inhalation. However carbon monoxide intoxication can occur without visible soot in the airways.[3]
- Curling's ulcers may be seen in the stomach and duodenum.
- Serous pleural effusions are common. Lungs are congested and appear "cooked."
- Spleen and adrenals may be enlarged, congested, and softened.

Box 11.1 outlines an infamous case where correct interpretation of burns present on a dead body proved crucial in solving the crime.

Table 11.2 Differences between antemortem and postmortem burns

Feature	Antemortem burns	Postmortem burns
1. Line of redness	Present around the burned part.	Absent
2. Blisters	Contain serous fluid with proteins and chlorides. Base is red.	Contain fluid and air. No proteins and chlorides. Base is dry and hard.
3. Vital reaction	Present	Absent
4. Enzymatic activity (by histochemistry)	Present (time related): Tissue cathepsin – immediate. Serotonin – 10mts. Histamine – 20mts. Esterase and ATPase – 1 hour Acid phosphatase – 3 hours. Alkaline phosphatase – 4 hours	Absent

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Box 11.1 The Case of Alfred Rouse

On the evening of Nov 6, 1930, two young men walking along a country road near Northampton, England, were surprised to see a neatly dressed man carrying a small case emerge from a ditch alongside the road. After the man had passed them and gone a distance ahead, he turned and shouted back to them, "It looks as though someone is having a bonfire up there." Just after he spoke, the men noticed flames on the horizon. When they reached the site of the fire, they discovered an automobile so consumed in flames that it was impossible to tell if there had been any occupants.

The police arrived and extinguished the blaze, and the charred remains of a person were found in the front seat. The flames had all but destroyed the car, but the license plate still was legible. When police traced the number, they discovered that the car was owned by one Alfred Arthur Rouse, a travelling salesman who lived in Finchley, North London. Rouse's wife, when contacted, said she had no idea where her husband could be located.

Rouse returned to London on Nov 7, and was taken in for questioning by the police. He claimed that he had given a ride to a man and when he had left the car briefly to relieve himself, the man had accidentally set fire to a container of petrol in the car. Rouse claimed further that he had tried to pull the man from the car, but was prevented by the intensity of the fire.

A police investigation revealed that Rouse was a notorious womaniser and bigamist. He himself admitted that between Nov 3 and Nov 7 he had been with Phyllis Jenkins, a young Welsh woman who was pregnant with his child. Because of his philandering and the increasing financial strain created by orders for child support, police surmised that Rouse had torched the car himself with the unidentified man inside in an attempt to escape his problems, hoping the victim would be mistaken for himself. Police formally arrested Rouse and charged him with murder.

Rouse was tried in January 1931. The prosecution demonstrated that the car's carburetor had been tampered with, thus enhancing their case that Rouse had planned the murder.

The Crown alleged that on the night of 5th November, 1930 (Guy Fawkes night), the accused met a man (whose identity was never established), gave him a lift in his motor car, and stopped in a small country lane near Northampton. It was alleged that he then struck his passenger on the head with a mallet and rendered him unconscious, placed him on the front seat of the car in a prone position and then poured petrol over him and set the car alight. The defence contended that the fire had arisen accidentally. The accused stated that he stopped the car on the roadside, and, as he noticed that his petrol was running low, he asked his passenger to fill the tank from a spare petrol can which he opened himself and placed on the driver's seat. He then walked down the road for a short distance, and when he turned round he saw that the car was in flames.

The bodywork of the car was completely burned out and only the chassis remained. The charred body of the deceased was found across the front seat of the car. The head was on the driver's seat, face downwards, and the body was on the passenger's seat. The left lower limb was bent up underneath the body with the leg flexed on the thigh and the thigh flexed on the abdomen. The right thigh was extended and the right leg was burned off below the knee. The defence maintained that this position of the body suggested that the deceased had been trapped in the blazing car and had made a violent attempt to get to the off-side door of the car. The medical witnesses called by the prosecution said that the position in which the body was found was consistent with the Crown's allegation. They explained that the muscles of the limbs had undergone heat rigor, and that although the left leg and thigh were free to bend as the limb projected beyond the edge of the seat, the right thigh could not become flexed because it was in contact with the seat. The accused was found guilty and sentenced to death. He was hanged at Bedford on Mar. 10, 1931.

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EFFECTS OF HIGH ENVIRONMENTAL TEMPERATURE

When the human body is exposed to high environmental temperature for a prolonged time, the following effects are seen :

1. Heat cramps.
2. Heat exhaustion.
3. Heat stroke.

HEAT CRAMPS

These occur in persons who are working in a hot atmosphere, and perspire profusely, for instance, manual labourers, firemen, miners, and stokers (hence the synonyms **fireman's** or **miner's** or **stoker's cramps**). They are characterised by painful cramps of the voluntary muscles. Profuse perspiration results in substantial loss of water and sodium chloride from the system, which accounts for the cramps. Treatment consists of restoring fluid and salt intake.

HEAT EXHAUSTION

This occurs in persons who physically exert themselves for a long period in a hot and humid atmosphere. It results from dehydration and salt depletion. Heat exhaustion (also referred to as **heat collapse, heat prostration,** or **heat syncope**), is characterised by headache, pallor, profuse perspiration, vomiting, subnormal temperature, muscular weakness, and circulatory collapse. Urine becomes scanty. Treatment consists of administering fluids of glucose-saline with sodium bicarbonate. The condition is rarely fatal.

HEAT STROKE

This results from hyperpyrexia following exposure to excessive heat. There is a disturbance in the normal physiology of the heat regulating center in the hypothalamus. The victim first experiences headache, visual disturbances, nausea, vomiting and dizziness, and then suddenly becomes unconscious. The body is flushed and hot, with dry skin. The body temperature may go as high as 43°C. The pulse is feeble and rapid, and the respiration stertorous. Treatment involves removal of the patient's clothing, and immersion of the person (up to his neck) in a tap-water bath till the temperature comes down to 38°C. This condition (also referred to as **heat hyperpyrexia, sunstroke,** or **thermic fever**), is often fatal, if not treated in time.

Autopsy Findings: Most of the victims die from disseminated intravascular coagulation. The most striking autopsy findings are large areas of pulmonary haemorrhage. The brain is congested and oedematous, with petechial haemorrhages in the white matter. Such haemorrhages are also found elsewhere in other organs of the body. Microscopically, there are micro-thrombi in small blood vessels of many organs with small foci of necrosis of parenchymal cells. Hepatic failure at times contributes to the death. Skeletal muscles may demonstrate rhabdomyolysis.

EFFECTS OF LOW ENVIRONMENTAL TEMPERATURE

HYPOTHERMIA

This is usually defined as a condition in which the body's core temperature falls below 35°C. Exposure to cold, exhaustion, fatigue, lack of food, disease, injuries, drugs, or alcoholic intoxication are usually major causative or aggravating factors of such a condition.

There is a progressive decrease in the metabolic processes in all the organs, a lowered oxygen dissociation from oxyhaemoglobin and oxymyoglobin, and a diminished blood flow through organs and tissues. The mechanism for temperature regulation becomes progressively inoperative.

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However, hypothermia protects the central nervous system from the effects of ischaemia, and is an important cause of "apparent death" (**suspended animation**). The heart rate slows down, and the cardiac output falls. Cardiac fibrillation occurs at temperatures below 28°C. Treatment involves administration of pre-warmed glucose by intragastric drip, and antibiotic prophylaxis.

Local effects of hypothermia produce **frostbite** and **immersion foot (trench foot)**. Frostbite occurs at temperatures ranging from 5°C to 8°C. If this is continued for several hours, gangrene occurs.

Autopsy Findings : A distinctive bright-pink colour of the blood and tissues is said to be characteristic of non-dissociation of oxygen from haemoglobin. There may also be prominent patches of purple discolouration of the limbs.

LIGHTNING

When there is discharge of electricity between clouds, lightning occurs. When the charge jumps between cloud and earth, it is called *lightning stroke*. Lightning chooses the easiest path (not the shortest), and hence it sometimes takes a wandering, zigzag path.

When lightning strikes a person, a potential of 1000 million volts causes a 20,000 ampere current to pass through the body of the victim (direct effect). This induces lethal electrocution.[4] The current may then disperse over an area close to where the victim is standing (indirect effect). When the lightning flash strikes a nearby metallic object it may give rise to induced voltage. The atmospheric gases become heated to 20,000°C, and this heating of gases in the path of lightning causes an explosive expansion. Although the duration of explosion is for a fraction of a second, the blast effect can be devastating. Objects may be fragmented and flung away from the site of lightning.

Autopsy Findings:

External lesions mostly take the form of burns –

- *Linear burns*:They vary from 6-25 mm in width. They are linear in shape, and are found more commonly on moist surfaces of the skin, because moist skin offers less resistance than dry skin.
- *Arborescent burns* (Fig 11.4): There is formation of superficial, thin, irregular, tortuous markings on the skin. They resemble the branching pattern of a tree, hence the name. Sometimes the pattern resembles a fern leaf (**filigree burns**).
- *Surface burns*: These burns occur due to heating up of metallic objects worn or carried by the person. Some times the object becomes magnetised.This can be ascertained with the help of a pocket compass.[4]

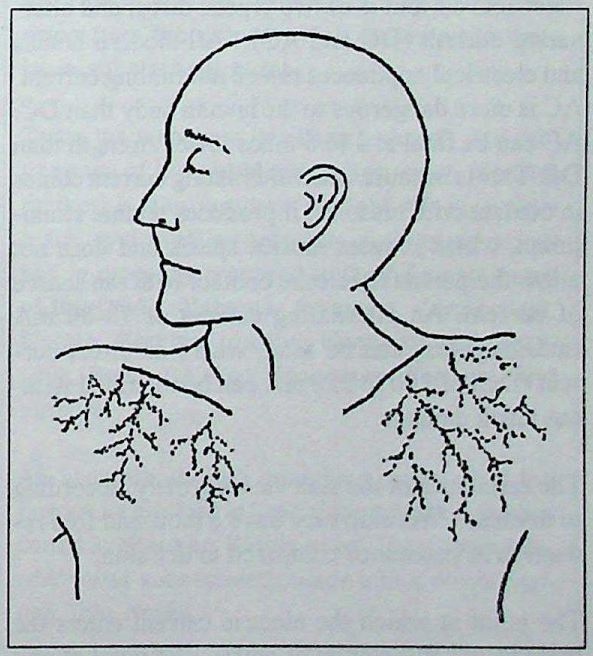

Fig 11.4

Other Effects –

- Clothes may be torn.
- Shoes may burst open.
- Rupture of tympanic membrane is often seen.[5]

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- Injuries from the skin may extend to involve subcutaneous tissue, muscle, and bone.
- Bony injuries may show periosteal elevation and destruction of superficial layers of the bone, or fractures.
- Petechial haemorrhags may be seen in the brain and the spinal cord. Chromatolysis and fragmentation of axons are common.
- Congestion of all organs, and pulmonary oedema are also common.

ELECTROCUTION

Injuries caused by electrocution respect all the known laws of physics and thus are predictable in their manifestations, under different physical conditions.

Electrical current is of two types : direct and alternating current (DC and AC). All modern homes and electrical appliances run on alternating current. AC is more dangerous to the human body than DC. AC can be fatal at 4 to 6 times lower strength than DC. This is because when alternating current comes in contact with muscles, it produces tetanic stimulation, which induces muscle spasm and does not allow the person to release contact with the source of current. An alternating current of 70-80 mA (milliamperes) can be fatal, where as direct current (DC) of 200 to 250 mA can be tolerated without much damage.

The resistance of the skin varies greatly, according to dryness. Wet skin may have a thousand fold reduction in resistance compared to dry skin.

The point at which the electric current enters the body is called the point of entry. The point of exit denotes the point from where the electric current leaves the body. Burns may be seen both at the point of entry as well as the point of exit (Fig 11.5 a and b). The electrical burn at the point of entry is called **Joule burn** or **endogenous burn**. It resembles the size and shape of the object causing it. Generally, it appears as a round or oval, shallow crater, surrounded by a ridge of skin, 2-4mm high. The floor is composed of pale, flattened skin with a surrounding hyperaemic zone.

The effects of electrocution vary depending on the point of entry and exit. If the point of entry is in one upper limb, and the earthing point is in the opposite foot, the current will pass across the chest in a diagonal direction and be most likely to produce myocardial fibrillation. Electrical current passing through the lower parts of the body may not produce fibrillation at all. Current passing through the head may not affect the heart, but can cause brainstem damage with central respiratory paralysis.[6]

Fig. 11.5a Electrical injury (entry)

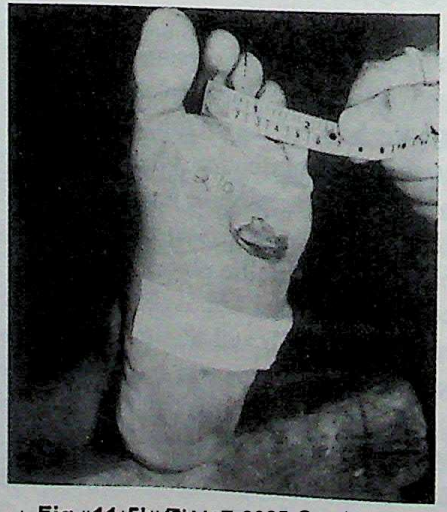

Fig. 11.5b Electrical injury (exit)

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Other types of burns

Spark burn is produced by intermittent contact with a live object, and is due to arcing of current from the conductor to the skin. A yellowish parchment-like skin surrounded by a pale area is seen. Sometimes thermal burns are seen over large surface areas of the body. While working with high - tension wire, etc, a person may be thrown off to a distance, yet may show different types of burns. This is also due to arcing. Occasionally there is actual charring of the skin with carbonisation. At times, there is confluence of spark burns resulting in a **crocodile skin** effect.

Causes of Death in Electrocution:

- Ventricular fibrillation
- Respiratory failure due to spasm of respiratory muscles.
- Cerebral anoxia.
- Delayed death due to complications of burns.

Autopsy Findings: Burns may be seen at the point of entry and exit. At the point of entry, the burn resembles the shape of the object causing electrocution. The skin may be ruptured at the point of exit.

All the organs are congested. Petechial haemorrhages are seen on serous surfaces. There may be pulmonary oedema.

Electrocution is by and large accidental. However, there are cases of electrocution due to suicidal and homicidal intentions. Judicial electrocution is practiced in some states of the U.S.A. (**Box 11.2**)

Box 11.2 Judicial Electrocution[7]

The use of judicial electrocution as a means of executing a sentence of death against convicted criminals was first mooted in the mid 19th century in New York, USA, and arose out of concerns as to the unpredictability of hanging in causing a quick, certain death. The first execution by electric chair took place in New York in 1890. A 1400 volt, 150 Hz AC current was applied to a prisoner for 17 seconds. The attending medical officer was so certain that this was sufficient, that he declared the person dead immediately. Unfortunately, within the next few minutes, the prisoner began to groan and started to breathe once again. The current was hastily reapplied for an additional 2 minutes. This time around, the prisoner was indubitably dead. Eye witnesses even reported that the electric current was so intense that they saw smoke rising from the corpse which appeared to have been substantially burnt.

Today the technique has been "refined", and proponents of judicial electrocution claim that the method is one hundred percent effective, very quick, and relatively painless. Judicial electrocution is presently practised in the following states of the USA : Alabama, Arkansas, Connecticut, Florida, Georgia, Indiana, Kentucky, Nebraska, Ohio, South Carolina, Tennessee, Vermont, and Virginia.

An accurate, though gruesome, account of the procedure involved in judicial electrocution is mentioned in **Stephen King**'s novel *The Green Mile*, which was subsequently made into a movie starring Tom Hanks.

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CHAPTER - 12

Medicolegal Aspects of Injuries and Death

"The law may be an ass, but more often it makes an ass out of those who try to circumvent it."

- Justice John Underwood

INJURIES AND THE DOCTOR

Since the nature of infliction of mechanical injuries is either accidental, suicidal, or homicidal, they are always associated with legal implications. Many such cases find their way to the criminal court. Therefore a doctor who has examined and treated such cases will inevitably be called to give evidence as an *expert witness*. His opinion is often crucial to the final outcome. It is imperative that every doctor be familiar with the procedure of examination and recording of data. The following constitute some important points in this regard:

- Every injury must be accurately documented as to the type, location, directions, dimensions, presence or absence of foreign bodies, time of infliction, etc. Measurements must be exact, and terms such as 'about', 'approximately', etc., must not be used.[1]
- As far as possible take photographs, or draw simple sketches of injuries.
- While the primary duty of a doctor is to treat a patient, he must never neglect medicolegal requirements.
- When a *wound certificate* is required by an injured victim, it is essential to insist on an official requisition from the police or court. In such a case, a doctor-patient relationship is not necessarily established, and there is only a doctor-subject relationship.
- An informed written consent is a must for any wound examination. But if the person is under arrest or if there is an order from court, the doctor can proceed with the examination even in the absence of consent. In fact, he is empowered "to use such force as is reasonably necessary for that purpose" (**Section 53 Cr.P.C**). However, whenever a female is to be examined by such force, "the examination shall be made only by, or under supervision of a female registered medical practitioner."
- The doctor has to exercise maximum skill and care during management. Required investigations have to be undertaken. Dying declaration, if required, should be recorded in the usual manner.
- It is desirable for a Government doctor to intimate to the police, all wounded cases, irrespective of the manner and nature of occurrence. But

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a private practitioner can abstain from such intimation in suicidal and accidental cases, if the patient does not give consent for such intimation.

- As per **Section 39 Cr.P.C**, every citizen (including a doctor) has to give information about certain offences to the police. Such offences include *murder* (**Section 300, IPC**), *culpable homicide* (**Section 299 IPC**), and *theft*, after preparation was made for causing death or hurt in order to commit theft (**Section 382 IPC**).

- The duty of the doctor while examining a wounded subject is to record the wounds in detail and give necessary opinion. Recording and subsequent issue of the wound certificate is a must. The findings should be kept confidential. The wound certificate must be prepared in triplicate, under the seal and signature of the Medical Officer who prepared it. The original as well as one copy must be handed over to the Police Officer concerned in a sealed, addressed cover. The other copy is to be retained by the medical officer.

The proforma of wound certificate is given in **Box 12.1**.

INJURIES AND THE LAW

NATURE OF INJURY

The law is not concerned with explicit differences between the terms *hurt, trauma, injury,* and *wound.* However, in strict legal parlance the word **injury** denotes any harm, illegally caused to any person in body, mind, reputation or property (**Section 44 IPC**). **Hurt** is defined as any bodily pain, disease, or infirmity caused to a person (**Section 319 IPC**). There is a special category of hurt referred to as **grievous hurt (Section 320 IPC)**, of which the following constitute examples:

- Emasculation.
- Permanent privation of the sight of either eye.
- Permanent privation of the hearing of either ear.
- Privation of any member or joint.
- Destruction or permanent impairment of the powers of any member or joint.
- Permanent disfiguration of the head or face.
- Fracture or dislocation of a bone or tooth.
- Any hurt which endangers life, or which causes the sufferer to be in severe bodily pain, or unable to follow his ordinary pursuits, during the space of twenty days.

Note:

- Self inflicted injuries are not covered by this definition.
- Emasculation means deprivation of masculine vigour.
- A crack fracture of bone which does not involve the medullary cavity will not be considered as fracture.[2]
- A mere stay in hospital for 20 days or more, will not constitute grievous hurt. It must be proved that during this period, he was in severe bodily pain or unable to follow his ordinary pursuits.
- An injury may be construed as "simple" from the medical point of view, but legally, it may fit one of the clauses (examples) under **Section 320 IPC**. The opinion of the doctor as to the nature of injury must always be as per legal definition.
- The final opinion as to whether a particular injury is simple or grievous is left to the discretion of the court. The role of the doctor is only to guide and help the court in deciding the issue.

Voluntarily causing hurt (Section 321 IPC) - Whoever does any act with the intention of causing hurt to any person, or with the knowledge that he is likely to cause hurt to any person, and does thereby cause hurt to any person, is said "voluntarily to cause hurt".

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Box 12.1 Wound Certificate

From

Dr. .....
.....
.....
.....

To

The Sub Inspector of Police
.....
.....
.....

Sir,

Sub: Issuance of wound certificate reg.
Ref: Requisition No. ....., dt. .....

Name of individual:
Sex:
Age:
Address:
Identification Marks : 1.....
2.....
Consent (*signature or thumb impression*)
Accompanying P.C.No., P.S.:
Brief History:
Date of Examination:
Time of Examination:
Beginning.....
Conclusion.....
Place:
Injuries:

Nature	Size	Site	Simple/ Grievous	Nature of Weapon	Dangerous Weapon or not	Additional Remarks

Signature of medical officer
Date:
Name:
Designation:

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Voluntarily causing grievous hurt (Section 322 IPC) - Whoever voluntarily causes hurt, if the hurt which he intends to cause or knows himself to be likely to cause is grievous hurt, and if the hurt which he actually causes amounts to grievous hurt, is said to "voluntarily cause grievous hurt".

Punishment for voluntarily causing hurt (Section 323 IPC) - Imprisonment of either description for a term which may extend to one year, or with fine which may extend to one thousand rupees, or with both.

Voluntarily causing hurt by dangerous weapons or means (Section 324 IPC) – Whoever voluntarily causes hurt by means of any instrument for shooting, stabbing, or cutting, or any instrument which when used as a weapon of offence, is likely to cause death, shall be punished with imprisonment of either description for a term which may extend to three years, or with fine, or with both.

Punishment for voluntarily causing grievous hurt (Section 325 IPC) - Imprisonment of either description for a term which may extend to seven years, and shall also be liable to fine.

Voluntarily causing grievous hurt by dangerous weapons or means (Section 326 IPC) - Whoever voluntarily causes grievous hurt by means of any instrument for shooting, stabbing or cutting or any instrument which when used as a weapon of offence, is likely to cause death, shall be punished with imprisonment for life, or with imprisonment of either description for a term which may extend to ten years, and shall also be liable to fine.

Occasionally, a doctor may be required to assess the extent and severity of physical disability from injuries. Details regarding permanent total or partial disablement are detailed in **The Workmen's Compensation Act, 1923**.

MANNER OF INJURY

Injuries may be caused by human beings, animals or machines. Such infliction may be accidental or, if they have been caused by a human being, they could be suicidal or homicidal.

Homicide

The term "homicide" means causing the death of a person by another person.

Classification

1. Lawful
   a. Justifiable
   b. Excusable

2. Unlawful (Culpable)
   a. Amounting to murder
   b. Not amounting to murder

Lawful Homicide -

It refers to the causing of death of a person which is permissible or excusable by law. The former is called *justifiable homicide*, for e.g., judicial hanging. *Excusable homicide* is that type of lawful homicide which is caused unintentionally by an act done in good faith, or in self defence.

Culpable Homicide (Section 299 IPC) –

Whoever causes death by doing an act with the intention of causing death, or with intention of causing such bodily injury as is likely to cause death, or with the knowledge that he is likely by such act to cause death, commits the offence of culpable homicide.

Murder (Section 300 IPC): Except in the cases hereinafter mentioned, culpable homicide is murder, if:

Firstly - The act by which the death is caused is done with the intention of causing death, or

Secondly - If it is done with the intention of causing such bodily injury as the offender knows to be

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likely to cause the death of the person to whom the harm is caused, or

Thirdly - If it is done with the intention of causing bodily injury to any person, and the bodily injury intended to be inflicted is sufficient in the ordinary course of nature to cause death, and

Fourthly - If the person committing the act knows that it is so imminently dangerous that it must in all probability cause death or such bodily injury as is likely to cause death.

Exceptions

Culpable homicide is not murder if:

- the offender, whilst deprived of the power of self-control by grave and sudden provocation, causes the death of the person who was responsible for the provocation, or causes the death of any other person by mistake or accident.
- the offender, in the exercise (in good faith) of the right of private defence of person or property, exceeds the power given to him by law and causes death of the person without premeditation, and without any intention of doing more harm than is necessary for the purpose of such defence.
- the offender, being a public servant or aiding a public servant acting for the advancement of public justice, exceeds the powers given to him by law, and causes death by doing an act which he, in good faith, believes to be lawful and necessary for the due discharge of his duty.
- it is committed without premeditation in the heat of passion, in the course of a fight, without the offender's having taken undue advantage or acted in a cruel or unusual manner.
- the person whose death is caused, being above the age of eighteen years, suffers death or takes the risk of death with his own consent.
- the death of a person other than the intended victim was caused inadvertently (**Section 301 IPC**).

Punishment for murder (Section 302 IPC) - Death (capital punishment), or imprisonment for life, and shall also be liable to fine.

Punishment for murder by life-convict (Section 303 IPC) - Whoever, being under sentence of imprisonment for life, commits murder, shall be punished with death.

Punishment for culpable homicide not amounting to murder (Section 304 IPC) - Imprisonment for life, or imprisonment of either description for a term which may extend to ten years, and shall also be liable to fine.

Accident

An accidental death is one where there is no intention to cause death on oneself, or by oneself on another. Numerically, **road traffic accidents** account for the great majority of accidental deaths world wide.

The purpose of forensic examination in traffic accidents is to determine the nature and manner of causation of injuries on a pedestrian, or the occupant or rider of a vehicle. At times, it may be necessary to reconstruct the details of an accident on the basis of the pattern of injuries, especially if there are no eyewitnesses.

Pedestrian Injuries -

The distribution and severity of pedestrian injuries depend on the speed and momentum of the impacting vehicle.

Classification

1. Primary impact injuries
2. Secondary impact injuries
3. Tertiary impact injuries

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Primary impact injuries

These are produced by the initial contact with the vehicle, and may be in the form of abrasions, lacerations or contusions. Occasionally, patterned injuries are seen.

Often, both legs may get fractured in the region of impact with the bumper of the vehicle (**bumper fracture**). Direct impact on the thorax not only results in fracture, but also may rupture the aorta. Direct impact on the hips may cause fracture of the vertebral column and pelvis.

Secondary impact injuries

They are cased by subsequent contact of body with vehicle after the primary impact. They usually result from the victim being flung into the air after the primary impact and subsequently landing against the hood, windscreen, or roof of the vehicle. Head injuries are common.

Tertiary injuries

These injuries are caused by striking against objects or surfaces other than the vehicle, such as the kerb, road surface, or street poles. Grazed abrasions, flaying injuries, etc., due to being dragged on the road are common.

Injuries of vehicular occupants -

Injuries to driver and other occupants are mostly related to sudden deceleration due to collision, the commonest type of vehicular accident. The driver and front seat passenger often sustain lacerations over the face due to impact against the windscreen, and fracture or dislocation of hip due to hitting against the dashboard. Fractures and dislocations of upper and lower limbs are also common. A special type of injury seen in the driver is the **steering wheel impact injury**, which causes fractures of sternum and ribs, as well as contusion or even rupture of the heart or aorta. Hitting against the rim of the steering wheel may rupture liver, spleen, kidneys, or duodenum. Sudden hyper extension or flexion of the neck may result in fracture-dislocation of cervical spine and injuries of the spinal cord (**whiplash injury**, see *page no.143*).

In traffic accidents, apart from examination of injuries sustained, a doctor has to observe whether the driver or pedestrian was suffering from any disease or disability, or if there is evidence of intoxication, and if so, whether the accident could have been a result of that. Identification of the vehicle involved in a **hit and run** case may be assisted by looking for trace evidence that is likely to have been exchanged between the victim and vehicle at the time of impact (**Locard's principle of exchange**, see *page no.258*)

Suicide

Injuries may be self inflicted in order to commit suicide, or they may be fabricated to substantiate a false allegation of assault. In some suicidal deaths, tentative or hesitation cuts may be seen. Ordinarily, there will be only one major (fatal) wound. Preferred sites are those which encase vital organs or structures, and which are easily accessible.

AGE ESTIMATION OF INJURY

Before an attempt is made to estimate the age of an injury at autopsy, it must be first determined clearly as to whether it is an **antemortem** or **postmortem** injury, i.e., whether it was inflicted during life or after death. Evidence of vital reaction will be present in an antemortem injury, such as swelling, redness, leucocyte infiltration, pus formation, and process of repair. The longer the victim lives after sustaining an injury, the more evident will be the vital reaction. The quantity of blood lost also may help in deciding whether the injury was antemortem or not. Thus, the presence of a large pool of blood probably suggests that the injury was antemortem. Wound gaping, adherent blood clots, etc., also indicate antemortem nature. Evidence of spurt-

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ing of blood is an important indication of antemortem injuries because of arterial pressure.

Injuries occurring during the **perimortem** period may lead to difficulties. Perimortem period refers to the period between the initiation and completion of death process. Injuries inflicted during this period do not demonstrate clear cut antemortem findings.

The exact age of a wound is always difficult to deduce, but an approximate estimate can be done on the basis of reparative processes (see **Chapter 8**). Apart from evidence of vital reaction, *enzyme histochemistry* may help in estimating the age of wounds. Enzyme activity is increased towards the periphery of the injury, and decreased in the region of the central zone. The activity of the following enzymes is increased in the peripheral zone : adenosine triphosphatase (1 hour), aminopeptidase (2 hours), acid phosphatase (4 hours), and alkaline phosphatase (8 hours).

CONSEQUENCES OF INJURY

A wound may heal, or may get complicated by processes other than healing. Sometimes there are undesirable sequelae, as in the case of healed ureteric rupture causing cicatrization which can result in hydronephrosis. Keloid formation in scars can lead to permanent disfigurement.

The most serious consequence of an injury is of course the possibility of death. Causes of death from wounds may be primary or secondary.

Primary (Immediate) Causes of Death

1. **Haemorrhage:**
   Bleeding from an injury may be external or internal. A sudden loss of 1000 cc of blood can result in shock and death. If the loss is spread over longer duration, the symptoms are less severe. However, advanced age and pre-existing morbid conditions can predispose to death. Also, effusion of even small amount of blood in certain situations can cause immediate death. An example is cardiac tamponade.
   Bleeding occurring at a later stage due to infection, or due to dissolution of clot is called *secondary haemorrhage*.

2. **Injury to vital organs:**
   Severe injury to vital organs such as brain, heart, or lungs can cause death irrespective of blood loss. It is important to note that if the organ is diseased, even trivial trauma can result in fatal consequences.

3. **Neurogenic shock:**
   The nervous control of cardiac and vascular functions is regulated by autonomic nervous system, and involves an optimum balance of two mutually antagonistic sympathetic and parasympathetic divisions. The former stimulates the cardiovascular system, while the latter exerts a tonic and inhibitory control over it, which is conservative-restorative in nature. The functioning of the cardiovascular system may be reflexly affected through the autonomic nervous system by factors such as emotion, variations in pressure in the carotid sinus, changes in the gaseous concentrations of blood, and excitation of peripheral afferent somatic or visceral nerves. These can cause either sympathetic stimulation or parasympathetic inhibition of cardiac function.

   - *Emotion*: Psychic shock may lead to parasympathetic inhibitory effects, while fear and pain may lead to sympathetic stimulation.
   - *Variations in pressure in the carotid sinus*: An increase in tension may lead to strong parasympathetic stimulation.

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- *Gaseous concentrations in the blood*: Low oxygen and high carbon dioxide may cause sympathetic stimulation, acting directly on medullary centres through chemoreceptors of the aortic and carotid bodies.

- *Excitation of peripheral nerves*: Stimulation of pressor and depressor afferent fibres can lead to either generalised vasoconstriction with raised blood pressure, or generalised vasodilatation with lowering of blood pressure. Weak stimuli cause depressor effects, while strong stimuli cause pressor effects. Severe pain as in squeezing the testicles, is an example of a strong stimulus.

- *Reflex autonomic stimulation*: Can lead to either sympathetic acceleration of pulse, myocardial contractility, generalised vasoconstriction and increased blood pressure, or prasympathetic reduction of pulse rate, myocardial contractility and central venous pressure, generalised vasodilatation and decreased blood pressure.

- *Air embolism*: Injury to the veins in the neck or chest can lead to air embolism. Air is sucked in due to negative venous pressure. An air lock in the right chamber of the heart results in acute circulatory block.

Secondary (Delayed) Causes of Death

1. **Infection**:
   Various microorganisms can infect wounds. Prominent among them are bacteria such as *staphylococci, streptococci, pseudomonas, clostridium tetani,* and *clostridium welchii*. Cellulitis, abscess, gangrene, pyaemia, septicaemia, and toxaemia are the sequelae. Infection of the original injury with breakdown of sutures or blood clot can result in secondary haemorrhage.[3]

2. **Thrombo-embolism**:
   Emboli and detached thrombi in injured veins can enter the right ventricle and (if large), can block the main pulmonary artery, or (if smaller), its branches. This can lead to cor pulmonale or pulmonary infarction. The time of occurrence is ordinarily 7-10 days after injury. Leg vein thrombosis is a complication of injuries to lower limb leading to confinement in bed.

3. **Fat embolism**:
   Fat can enter damaged veins at the site of injury from fractured long bones, or crushed fatty tissue. After passing through the heart it can enter the systemic circulation. Fat embolism may manifest as punctate haemorrhages in the brain or skin. Oedematous fluid from cut surfaces of lungs may contain fat globules. Urine may also contain fat globules.

4. **Crush syndrome**:
   Crushing of muscles can liberate haeme and myoglobin, which enter the circulation and get excreted through the kidneys. This can lead to acute tubular necrosis. Death may occur in a week's time. At autopsy, the kidneys appear large and bulky, and haeme casts may be found in the distal convoluted tubules.

5. **Other causes**:
   - Trauma can exacerbate an existing disease, e.g., tuberculosis.
   - Death can occur due to trauma to a diseased organ, e.g., malarial spleen.
   - Death can also occur from complications arising out of an injury, e.g., hypostatic pneumonia in bed ridden victims of spinal cord injury.

INJURIES AND WEAPONS

The Indian Penal Code describes two types of weapons:

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Deadly weapon (Section 148 IPC) : Whoever is guilty of rioting, being armed with deadly weapon, which when used as a weapon of offence is likely to cause death, shall be punished with imprisonment of either description for a term which may extend to three years, or with fine, or with both.

Dangerous weapon (Section 324 & 326 IPC) : Described on *page no. 158*

Weapons can also be classified as being blunt, sharp, or pointed. They are further sub-divided into light and heavy weapons. The lightness or heaviness of a weapon is based on subjective opinion, since no optimum weight is prescribed by law for such classification.

INJURIES AND VOLITIONAL ACTS

The ability of a person to move and perform certain actions after sustaining an injury, is a matter of controversy in several cases. Expert medical opinion may be invited to clarify such cases. The reaction to an injury varies from person to person. Dogmatic statements can never be given. The medical officer must be very guarded in his opinion.

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CHAPTER - 13

Asphyxial Deaths

"The right to breathe clean, unpolluted air is the most fundamental of human rights. You stop breathing this, you stop living".

- **Sylvester Dias** (Human Rights Activist)

Literally, the term **asphyxia** denotes absence of pulsation (*pulselessness*), though its usage in Forensic Medicine has generally come to mean a lack of oxygen.[1] Actually, asphyxia is best described as an interference with respiration due to any cause – mechanical, environmental, or toxic. The term **anoxia** refers to a lack of oxygen.

CAUSES OF ASPHYXIA (Fig 13.1)

- Closure of external respiratory orifices : **smothering**.
- Occlusion of air passage by pressure on the neck: **hanging, strangulation,** or **throttling**.
- Occlusion of air passage from within : **choking**.
- Lack of oxygen in the atmosphere, or inhalation of irrespirable gases: **suffocation**.
- Restriction of respiratory movements of the chest or abdomen : **traumatic asphyxia**.
- Prevention of gas exchange in the lung by fluids : **drowning**.
- Inability to utilize oxygen by peripheral tissues: **poisoning** (e.g., cyanide).

SIGNS OF ASPHYXIA (Fig 13.2)

I. CLASSICAL SIGNS[2]

1. **Oedema and congestion of organs:**
When the neck is compressed (as in hanging or strangulation), the venous return gets obstructed. This leads to congestion, i.e., the face, lips, and tongue become swollen and red. Sometimes there is darkening due to cyanosis (*vide infra*).

Congestion is associated with tissue swelling if there is continued venous obstruction. When circulation stops completely with ensuing anoxia, the walls of capillaries become permeable resulting in exudation of fluid from the capillaries into neighbouring tissues, leading to oedema.

2. **Petechial haemorrhages:**
Due to the impaired integrity of capillary walls, red blood cells escape through, producing small bleeding points, varying from pinpoint to pinhead size. These are known as petechial haemorrhages or petechiae or **Tardieu spots**. They are also attributed to rupture of venules, and are most commonly seen in the sclera, conjunctiva, or over serous membranes such as pleura and pericardium. If the bleeding spots are larger than 2 mm, they are called ecchymoses.

3. **Cyanosis:**
The colour of oxygenated blood is normally scarlet red. When haemoglobin is not fully saturated with oxygen, i.e., it is reduced, the

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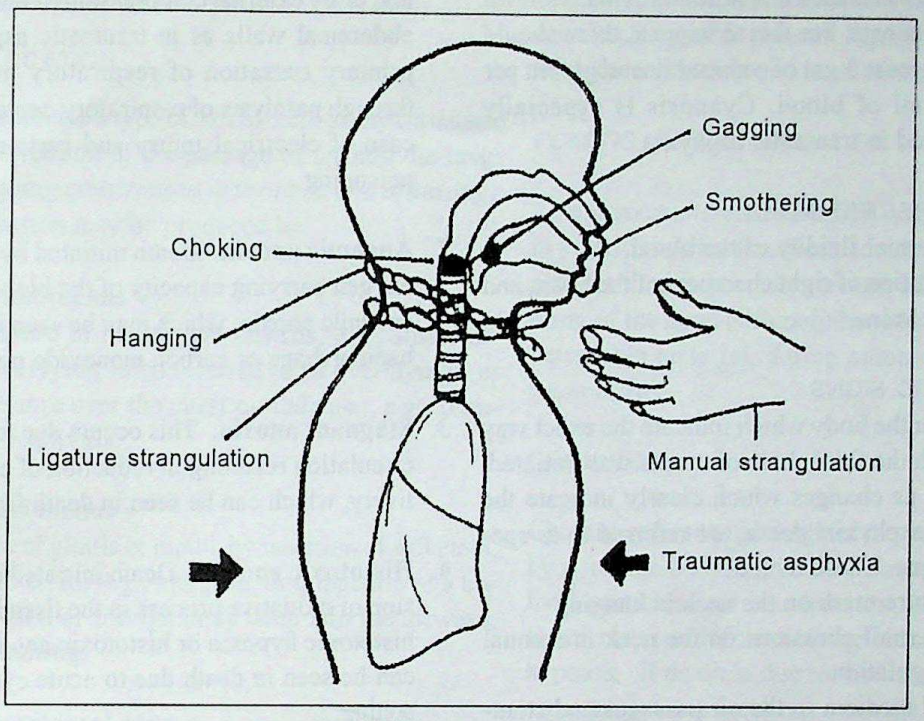

Fig. 13.1 Types of asphyxia

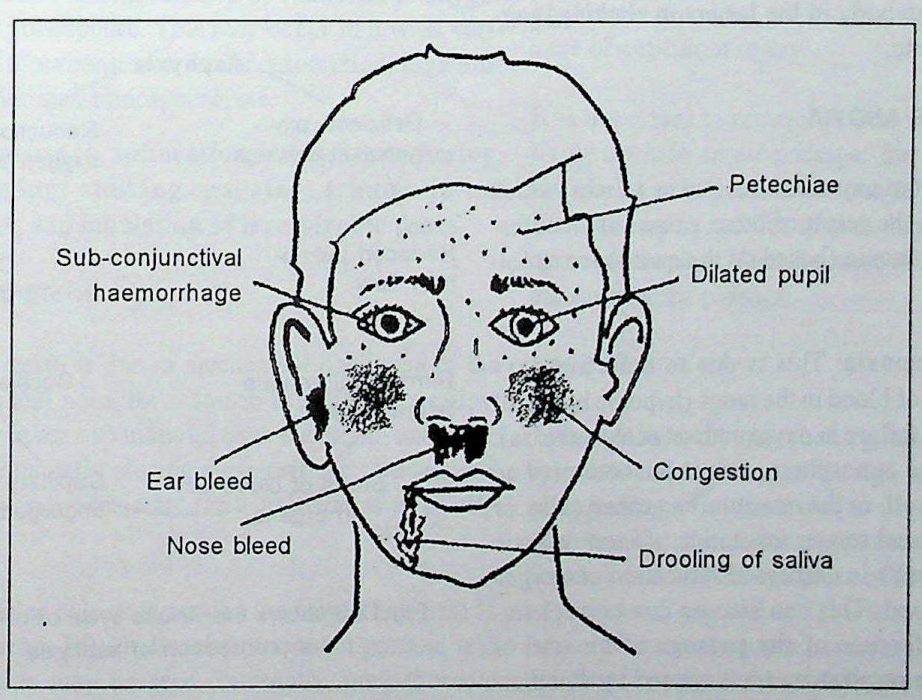

Fig. 13.2 Signs of asphyxia

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blood assumes a bluish colour. This is known as cyanosis. For this to happen, there should be at least 5 gm of reduced haemoglobin per 100 ml of blood. Cyanosis is especially marked in traumatic asphyxia.

II. NON-SPECIFIC SIGNS

1. Abnormal fluidity of the blood.
2. Dilatation of right chambers of the heart, and great veins.

III. SPECIFIC SIGNS

Signs on the body which indicate the exact way in which the fatal chain of events was initiated, as well as changes which clearly indicate the type of asphyxial death, are referred to as specific signs. These include:

- Ligature mark on the neck in hanging
- Fingernail abrasions on the neck in manual strangulation.
- Fluid medium in the air passages and stomach in drowning
- Foreign body in the larynx in choking, gagging, etc.

CAUSES OF ANOXIA

Asphyxia from any cause results in anoxia, which may also be the result of other causes. The different types of anoxia (based on the cause) are as follows[3]:

1. **Anoxic anoxia**: This is due to defective oxygenation of blood in the lungs (hypoxic hypoxia) or a total failure in oxygenation (anoxic anoxia). In vitiated atmosphere such as the bottom of an unused well, or the interior of a granary, silo, or underground sewer, inhalation of inert or toxic gases results in decreased saturation of oxygen in the blood. This can also be due to mechanical interference of the passage of air into or down the respiratory tract caused by smothering, choking, throttling, strangulation, and hanging, or by external compression of the chest and abdominal walls as in traumatic asphyxia, or primary cessation of respiratory movements through paralysis of respiratory center as in the case of electrical injury and certain kinds of poisoning.

2. **Anaemic anoxia**: Death initiated by a reduced oxygen carrying capacity of the blood is called anaemic anoxia, which may be seen in massive haemorrhage or carbon monoxide poisoning.

3. **Stagnant anoxia**: This occurs due to impaired circulation resulting in reduction of oxygen delivery, which can be seen in death from shock.

4. **Histotoxic anoxia**: Death initiated by depression of oxidative process in the tissues is called histotoxic hypoxia or histotoxic anoxia, which can be seen in death due to acute cyanide poisoning.

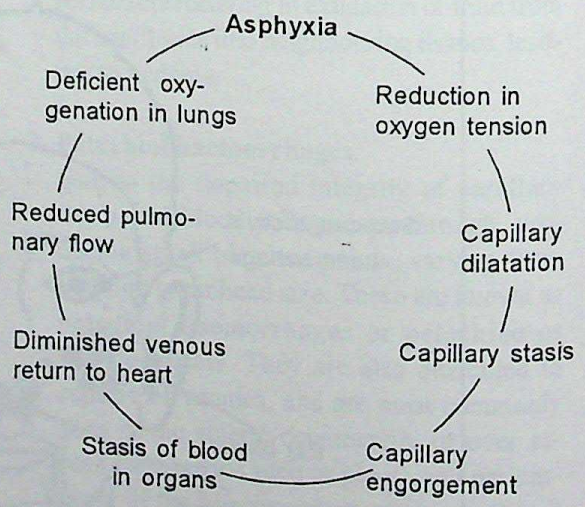

Fig 13.3 shows the vicious cycle of events in the progression of asphyxia

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MECHANICAL ASPHYXIA

SUFFOCATION

Suffocation is a type of mechanical asphyxia caused by obstruction of the passage of air into the lungs (*excluding constriction of the neck and drowning*). Suffocation may be produced by:

External means -
By closure of mouth and nostrils, e.g., **smothering, overlying** (also referred to as overlaying), or by pressure over the chest or abdomen, e.g., **traumatic asphyxia**.

Internal means -
Closure of glottis or mouth by insertion of soft cloth, paper, etc., for e.g., **gagging**, or impaction of a bolus of food or foreign body deep into the airways, e.g., **choking**.

Environmental causes -
This refers to death caused by reduction of oxygen in the atmosphere. This may occur in a wide variety of situations such as decompression, high altitude, vitiated atmosphere, etc.

To summarise, **suffocation** includes **smothering, overlying, choking, gagging, traumatic asphyxia**, and **inhalation of irrespirable gases**.

SMOTHERING

Here, death is due to mechanical occlusion of the mouth and nose, for instance by pressing a pillow or any other soft material over the mouth and nose, or by manually closing these orifices. Infants and elderly individuals are the usual victims of smothering.

A circular pale area around the mouth and nose is often seen. Bluish discolouration, congestion, and petechiae may be seen. Internally, intra-thoracic petechiae are common.

Scratches and nail marks on the face of the victim are also frequently present. Lips, gums, and tongue may show bruising and lacerations.

PLASTIC BAG SUFFOCATION

This is encountered in children who put their heads in plastic bags for fun. Such accidental deaths have also resulted in sexually deviant adults due to covering of the head with polythene sheet or other impermeable material, during autoerotic sexual acts (*vide infra*).

CHOKING

Choking refers to blockage of the trachea or larynx by a foreign body, e.g., coins, dentures, fish bones, food particles, seeds, aspiration of vomitus, etc. Death may result from neurogenic cardiac arrest or hypoxia. If death is due to the latter, all the usual signs such as congestion, cyanosis, and petechiae may be present at autopsy. Another example of choking is the entry of blood into the airways in a case of cut-throat injury.

It is important to remember that in order to effectively occlude an air passage, the object need not always be as large as the lumen of the air passage. Even an object smaller in size than the lumen can cause a complete block because of reflex spasm of the respiratory passage.

CAFE CORONARY

Although by and large, choking is simple mechanical obstruction, entry of foreign material may in some cases cause death due to vagal inhibition. A person under the influence of alcohol may sometimes choke over a large piece of meat or bone, due to depressed gag reflex. The foreign body in the larynx or trachea causes parasympathetic stimulation from laryngeal nerve endings, which results in cardiac arrest and death. This term is actually a

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misnomer, and was the result of such cases being earlier attributed (mistakenly) to myocardial infarction.

GAGGING

This is strictly speaking, a form of choking. The usual situation arises from a soft cloth being thrust into the mouth of a person to render him incapable of shouting. Even if the cloth happens to be small in size, it partially blocks the air passage, soaks up saliva, and swells. Due to constant foreign body irritation, there is pooling of saliva and death subsequently occurs due to obstruction of air passages. As is evident from above, it may be accidental when done to render a person speechless in the course of the commission of a crime. It may however be homicidal in infants.

CAUL BIRTH [5]

When a child is born with its head covered by the amniotic sac, it is likely to die of suffocation unless the membranes are incised at birth, and the child's air passages cleaned. This is called caul birth.

TRAUMATIC ASPHYXIA

Synonyms : Crush asphyxia, Compression asphyxia In this case, death results from pressure and fixation of the chest and/or abdomen. Thus if a person is crushed under a vehicle or in a house collapse, or an earthquake, or in a stampede, the result is traumatic asphyxia. The steering wheel of a motor car violently impinging on the driver's chest in a car accident, is also an example of traumatic asphyxia. Because of the pressure on the chest, the victim is unable to breathe and dies of asphyxia. As would be evident from the foregoing, traumatic asphyxia is mostly an accidental phenomenon.

Autopsy findings: Gross congestion, cyanosis, and petechiae of the face, neck, and shoulders are the most remarkable findings. There is often a very dark discolouration of the body above the level of constriction. Instead of petechiae, haemorrhages are frequently seen in the conjunctivae. Copious bleeding may occur through the ears and nostrils. There may be injuries resulting from the heavy object pinning the chest. Internally, subpleural haemorrhages and Tardieu's spots are observed. The right heart and all the veins above the atria are markedly distended. The blood is forced back into the great veins due to pressure on the chest. As the venous valves in the subclavian vessels prevent displacement into arms, the extra volume is forced up the valveless jugular system to cause congestion of the head and the neck. All the appearances of asphyxia (external and internal), are intensely pronounced.

Injuries to the chest, including fractures of the ribs and lacerations of the lungs and heart may be seen. If ribs are fractured, they are usually bilateral, multiple, and involve the angles of the ribs. In a steering wheel impact, there may be a transverse fracture of the sternum at the junction of the manubrium with the body (*buckled sternum*).

OVERLYING

This occurs when a parent or some other adult shares the same bed with an infant. During sleep, he/she may inadvertently roll over the infant asphyxiating it. Along with petechial haemorrhages, there may be evidence of flattening of the nose and face. A clear pattern of the object (fabric) involved in compression, is sometimes seen on the infant's cheek or body. There may be bloodstained froth at the mouth and nose.

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BURKING [6]

This term is derived from the infamous criminal **Burke**, who with his accomplice **Hare**, killed old people (mostly beggars and tramps) by a combination of smothering and traumatic asphyxia, and sold the bodies to the medical school in Edinburgh, (**Box 13.1**).

HANGING

Definition

Hanging results from constriction of the neck as a result of suspension of the body by a ligature, where the constricting force is the weight of the body or a part of the body. Hanging should always raise the presumption of suicide, while strangulation (*vide infra*) must always be considered homicidal unless proved otherwise. In India, hanging is among the top 5 methods of choice for committing suicide, the other preferred methods being poisoning, drowning, and burning.[7]

Causes of death

Immediate causes

- Prevention of entry of oxygen into respiratory passages (airway occlusion).
- Vascular congestion from compression of large veins, subsequently interfering with blood supply to the brain leading to anoxia.
- Combination of the above.
- Compression of arteries of the neck with direct anoxic consequences on the brain.
- Sudden cardiac arrest due to reflex vagal inhibition.

Delayed causes

- Infection.
- Sequelae of hypoxia to the brain.
- Oedema of the organs, especially the lungs.

Mechanism

A constricting pressure of just 2 kgs/m² is sufficient to compress the jugular venous system leading to intracranial venous congestion. The victim loses consciousness gradually and the inexorable build up of venous engorgement produces cerebral oedema, anoxia, and eventually death.

A constricting pressure of 5 kgs/m² obliterates both carotid arteries. The victim loses consciousness rapidly, and the abrupt cessation of blood to the brain results in cerebral anoxia. These mechanisms explain why suicide victims may succumb even in cases of **partial hanging**, where the body does not hang free of the ground. In cases of **complete hanging**, where the entire body weight is suspended on the ligature and no part of the body touches the ground, there is immediate occlusion of air passage along with obliteration of great vessels in the neck. Airway occlusion can be the result of direct compression of the larynx or trachea, or alternatively, the ligature may force the base of the tongue upward and backward against the posterior pharyngeal wall, pushing the soft palate upward and depressing the epiglottis over the laryngeal opening. The pressure needed to occlude the trachea is 15 kgs/m².

Points of importance

- Pressure on the baroreceptors situated in the carotid sinus, the carotid sheaths, and the carotid body can result in slowing of the heart or cardiac arrest. The impulses travel to the 10th cranial nerve nucleus through the glossopharyngeal nerve, and return via the vagus nerve to inhibit the heart.

- In every case of hanging, the scene of the incident should be inspected with reference to the type of platform, evidence of stains, signs of

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struggle, presence of suicide note, or pornographic material (in autoerotic sexual asphyxia). The body should be examined for injuries, type of clothing, and the nature of ligature mark.

- The ligature material used could be anything from a bed sheet to a leather belt, wire, rope, or even a handkerchief, as in most cases there is little premeditation. The length, width, and pattern of the material should be noted. The most important point is matching of ligature material with the ligature mark. The ligature material should always be cut away from the knot, and preserved for later laboratory examination.

- Hanging could be accomplished even without the use of a noose (either fixed or running). For example, suspension could occur from a steering wheel in the case of a vehicular accident, or from the rungs of a ladder when the person who is using it slips while climbing.

- An important point relating to the mode of application of ligature is that, a ligature knotted firmly following the first turn, and then knotted after the second turn is unlikely to be a suicide. The position of the knot in suicidal hanging is commonly over the occiput, or either side of the neck. The most common position is on the right side of the neck.

- Grooving of the ligature mark is due to congestion and associated oedema. These are generally more marked near the upper border of the mark. The ligature groove will be deepest on the opposite side of the knot when the noose is tied with a fixed knot. The point of suspension chosen by a suicide normally will be one that is within reach, with or without the aid of some platform.

Symptoms

A victim of hanging experiences simultaneously, ringing in the ears, blurring of vision, mental confusion, loss of power of thought, loss of consciousness, convulsions, and death.

Autopsy findings

Ligature mark

The ligature leaves a distinct furrow of its own width and pattern on the skin surface. The bed of the furrow may be pale, and the edges reddish-brown due to abrasion caused by the constriction. Sometimes the entire mark appears reddish-brown. Ecchymosis and congestion of adjacent skin may be seen. In general, the thinner and tougher the material used, the more pronounced is the ligature mark. Similarly, the softer and broader the material, the less distinct is the ligature mark. The skin in the region of the ligature mark is generally dry and hard.

The pattern of the ligature used often gets imprinted on the skin as a pressure abrasion. The ligature mark is usually situated above the level of the hyoid bone, and is oblique, passing backwards and upwards symmetrically on either side to the point of suspension. The mark is not seen at the point of suspension, and also in those regions where there is intervening hair or clothing. At times there may be more than one ligature mark when the material has been wound around the neck more than once. In such cases, the skin between the ligature marks will appear bruised due to pinching.

Microscopically, the ligature mark displays the usual characteristics of an abrasion, showing desquamation and flattening of cells of the epidermis. If death has occurred quickly, vital reaction may be quite difficult to demonstrate.

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The knot

In most cases of suicide by right-handed victims, the knot is situated on the right side because tying of the knot is easier. It may be a slipknot as is usual in suicidal hanging, or a granny knot. The common sites for the knot include the right or left side of the neck, and the occiput. Cases of suspension where the knot is below the chin, are rare. The groove is deepest opposite the position of the knot. When hanging is from a low point of suspension, the mark may be horizontal and could resemble strangulation, although in hanging it is invariably above the level of the thyroid cartilage.

Other external features

- The face may be congested, puffed up, and bluish.
- Bloody froth may be seen in the region of the mouth.
- Post mortem lividity is seen in the distal parts of upper and lower limbs, which gets fixed after 6-8 hours. This is because the hands and feet constitute the dependent parts of the body, in the suspended state. If the body remains suspended for a long time, petechial haemorrhages are seen on the lower limbs.
- The head is tilted away from the knot.
- The ligature may press on the salivary glands causing increased salivary secretion, which causes dribbling of saliva from the angle of the mouth. If the body is viewed later, dried up salivary stains running down from the angle of the mouth on to the front of the chest may be seen. This conclusively suggests the antemortem nature of hanging.
- The tongue is protruded and may have been bitten.
- If the knot presses on the cervical sympathetic ganglia, the eye on that side remains open, and the pupil is dilated. This is also a vital antemortem feature, and is often referred to as *Le facie sympathique*.

Neck dissection

Blood often extravasates into the subcutaneous tissues of the neck, beneath the ligature mark. Contusion of sternocleidomastoid and platysma may be present. The intima of the carotids may be ruptured transversely.

Occasionally, the hyoid bone is fractured. If such is the case, it is more common in the case of a deceased above 40 years of age, in whom the greater cornua have fused with the body.[8] As the hyoid bone is pressed backwards against the vertebrae, it results in an *anteroposterior compression fracture*. This is also called *abduction fracture*. The greater cornua therefore diverge outwards. The broken fragments of the bone also have an outward angulation and can be freely moved only outwards, but cannot be manipulated inwards. The periosteum is torn on the inner aspect of the fracture. Superior horn of thyroid cartilage may be found fractured in some cases.

The lymph nodes of the neck above and below the ligature mark, show evidence of congestion, stasis, and haemorrhage. This is never seen in postmortem hanging. In hanging associated with a long drop, destruction of larynx, and fracture-dislocation of cervical vertebrae are common. Punctate haemorrhages may be present in the laryngeal mucous membrance. However, cases have been reported where even a diseased and weakened cervical spine has not fractured in hanging.[9]

Internal features

All the usual signs of asphyxia are generally seen, (*page no.* 164).

MEDICO LEGAL ASPECTS

Hanging is invariably suicidal (Fig 13.4). Accidental and homicidal hangings are rare. Any material

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that is immediately available may be used as ligature material. A foot stool may be found at the scene of death, wherein the person attempting suicide ties one end of the ligature material to the beam or ceiling fan, stands on the stool, adjusts the noose around his neck and steps off the stool.

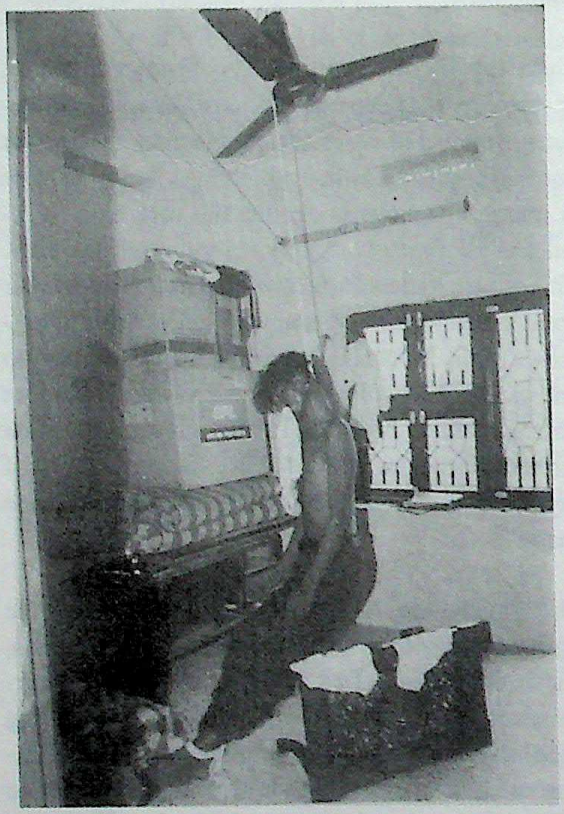

Fig 13.4 Suicidal hanging (simulating homicidal hanging)

When the feet do not touch the gound and the body is completely suspended by the ligature, it is called **complete hanging**. If the body is in a reclining or sitting position, or if the feet touch the ground, it is called **partial hanging** (Fig 13.5).

When the body is suspended by a ligature which runs upwards from either side of the thyroid cartilage, and the point of suspension is above the occiput, it is called called **typical hanging**. When the point of suspension is elsewhere such as the right or left side of the neck, it is called **atypical hanging** (Fig 13.6).

Although by definition hanging is defined as suspension of the body by a ligature, it may occur without the use of a ligature, e.g., being suspended from the rung of a ladder, if one slips while climbing.

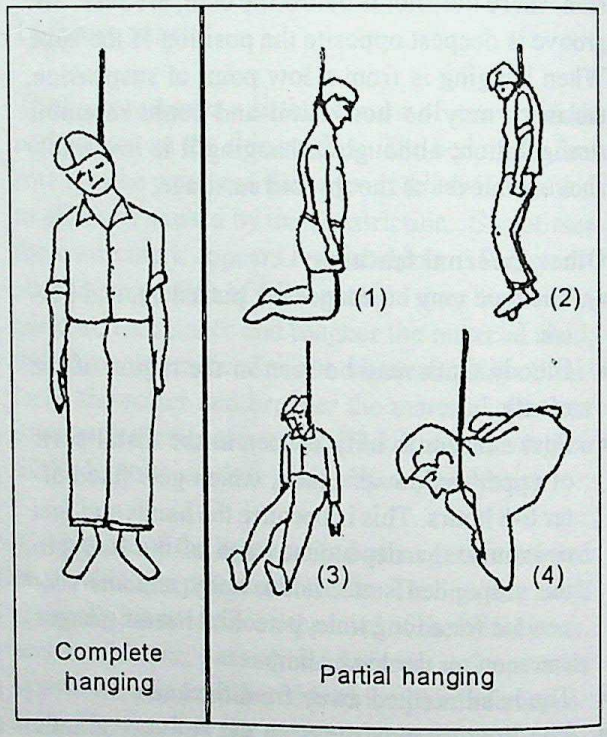

Fig. 13.5

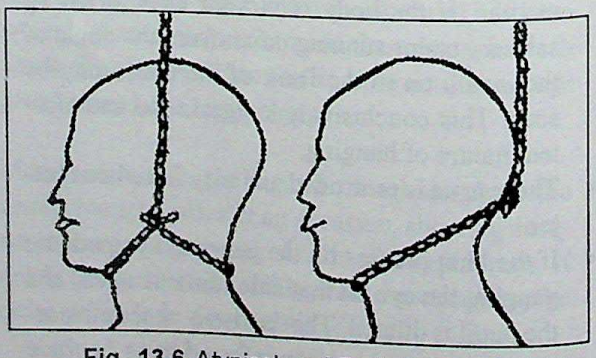

Fig. 13.6 Atypical and Typical hanging

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Homicidal hanging

This is difficult to accomplish unless the victim is a child, or a weak and feeble individual, or one who is weakened by alcohol or drugs.

Lynching * : It is a form of homicidal hanging where a person suspected to be involved in dacoity, murder, or rape, is overpowered by a mob, and hanged forcibly from a tree or lamp post.

Judicial hanging

In some countries (including India), hanging is the mode of legal execution of death sentence. The condemned man is first led to the gallows, and his face is covered with a black mask. The noose is adjusted around his neck. He is then made to stand on a platform, which by means of a lever is pulled back, so that he drops down to 5-7 metres. When he comes to the end of the rope, there is a violent jerk which causes a fracture dislocation of upper cervical vertebrae. This disrupts the brainstem resulting in instantaneous death.

Accidental hanging

Children playing with a ligature may sometimes accidentally hang themselves.

Postmortem hanging

Occasionally, after a victim has been murdered, the body may be suspended to simulate hanging, in order to mislead the police into believing it to be a case of suicide. Findings of asphyxia will not be prominent in such a case.

Auto erotic hanging

This is a rare type of hanging seen mostly in white males in Western countries. It is one form of sexual paraphilia. The individual (often dressed in female attire), partially asphyxiates himself with a ligature. The resulting partial anoxia is believed to enhance sexual pleasure by masturbation. The neck is usually protected by a soft cloth, so that the ligature mark is not seen. Some element of bondage (tying) is also seen in such auto erotic hanging cases. Pornographic literature may be seen scattered around. These cases invariably occur in secluded or private spots. In very rare cases, females have been involved in such deviant practices.[10]

STRANGULATION

His face is black and full of blood, his eyeballs farther out than when he lived,

Staring full ghastly, a strangled man.

His hair unreared, his nostrils stretched with struggling,

His hands abroad displayed as one that grasped and tugged for life,

And was by strength subdued.

- William Shakespeare

Definition

Strangulation refers to the application of external pressure on the neck either by bare hands, or by a ligature, or by any other material. It is a form of asphyxia caused by constriction of the neck without suspending the body. Strangulation that is effected by a ligature is called *ligature strangulation*, while that which is accomplished by bare hands is called *manual strangulation*, or *throttling*.

MANUAL STRANGULATION (THROTTLING)

When the hands are used to squeeze or compress

Named after **Capt.William Lynch of Virginia, USA, who was a law enforcement officer notorious for dispensing harsh justice in the form of on-the-spot hanging without trial.*

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Fig. 13.7a

Fig. 13.7b

Fig 13.7 (a) Throttling (b) Contusion underneath the skin in case of throttling

the neck, the resulting asphyxia is called manual strangulation or throttling. Occasionally, compression of carotid structures (e.g., carotid sinus), may result in cardiac arrerst. It is always homicidal. At times, a mild playful tweak on the neck has resulted in death from vagal inhibition.

Autopsy findings (Fig 13.7): Signs of asphyxia are seen. In addition, the following specific findings may be present:

Bruising of the neck: Bruising occurs due to the assailant's fingers grasping the neck. The bruises are usually circular, dark red or purple in colour, and are 1-2 cm in size. If the fingers slide over the skin, elongated marks may be seen.

Abrasions on the neck: Scratches may be caused by the fingernails of either the assailant or the victim. They may be curved or linear. During autopsy, scrapings from under the fingernails of the deceased, may be taken for DNA fingerprinting and compared with that of the suspect, because the victim may have scratched the assailant in an attempt to ward off the attack.

Internal appearances: The tissues of the neck are often markedly contused. Bleeding may be seen in the strap muscles or platysma. If excessive force has been applied, bleeding from larynx may have occurred.

Fractures of hyoid & thyroid (Fig 13.8): The hyoid bone calcifies, i.e., it becomes a single bone by the age of 40 to 45 years. In younger individuals, it is usually cartilaginous and hence fractures are uncommon. The thyroid horns are especially vulnerable to pressure on the neck. Antemortem fractures of the thyroid and hyoid are diagnosed by evidence of haemorrhage around the fracture site, with blood spots under the periosteum. The broken region may be slightly crepitant. Microscopically, there may be extravasation of RBCs.

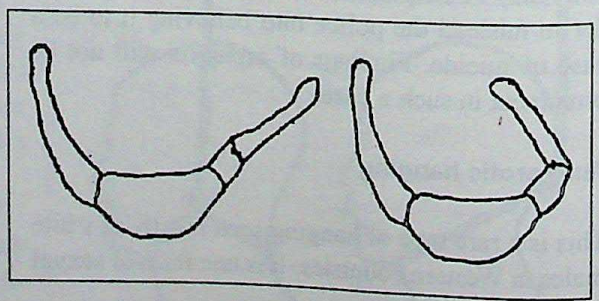

Fig 13.8 Antero-posterior (left) and inward compression fracture (right) of hyoid bone

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BANSDOLA

In this form of strangulation, a wooden pole or rod is placed over the front of, and another behind the neck. One end of these rods is fastened together by a rope. The other ends when forcefully brought together squeeze the neck to cause death.

MUGGING

It is brought about by squeezing the neck of the victim in the crook of the elbow, or the bend of the knee.

GARROTTING

This is said to be the method of judicial execution in some countries. The victim is made to wear a neck collar of metal. Death is brought about by tightening the collar. Garrotting may also be effected by an assailant who comes up behind a seated victim, throws a ligature around his neck, and pulls it tight. Since there is an upward pull, the resulting ligature mark may resemble that of hanging.

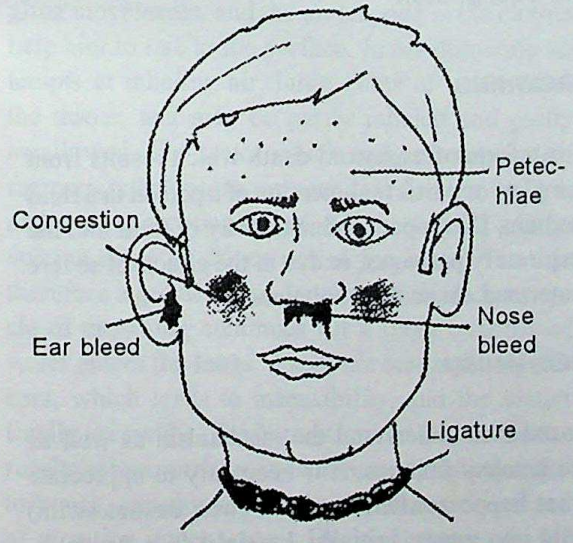

Fig 13.9 Strangulation

Material used for ligature strangulation can be anything from flexible rubber tubing to cloth, or sticks, wooden planks, belt, wire, rope, etc. In infants, homicidal strangulation may be brought about by winding the umbilical cord around the neck.

Pseudostrangulation [3]: In the neck of a dead (obese) person or infant, the skin folds may sometimes mimic ligature marks, which may be confused with strangulation marks. However, dissection reveals absence of bruising or abrasions.

AUTOPSY FINDINGS OF STRANGULATION

Classical and non-specific signs of asphyxia are prominent. Tongue is often protruded. Bleeding from the ears or the nose may be seen.

The **ligature mark** (Fig 13.10) is usually seen as a depression around the neck because of the oedema of the tissues above and below it. The skin over the mark is invariably dry and hard. The base of the mark appears pale, while the edges may show small abrasions. The width of the mark depends on the ligature used. When there is a pronounced pattern in the ligature material, the pattern may get imprinted on the skin as an imprint abrasion. The mark is usually situated around the middle of the neck at the level of the thyroid cartilage, in contrast to the higher location in hanging.

In most cases it runs horizontally, contrasting with the oblique direction in hanging. Sometimes, several turns may have been effected with the ligature, which is then secured with one or more knots. The ligature mark completely encircles the neck in strangulation, while in hanging it is absent near the point of suspension. The position of the knot is indicated by a wider impression on the skin.

The type of knot is generally a reef or granny knot. The occurrence of a narrow zone of engorgement or bruising immediately above and below the

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groove, confirms that the victim was alive at the time when the ligature was applied. In addition, abrasions may be present. Fingernail marks, sometimes linear and vertical may also be present, suggesting efforts on the part of the victim to ease the pressure of the ligature.

Even if putrefaction has set in, the ligature mark usually remains distinct. Asphyxial component is always more pronounced in strangulation, as compared to hanging.

Carotid arteries often show transverse intimal tears. Fracture of left or right superior horn of thyroid cartilage is a common feature. If more force is applied, there may also be fracture of cricoid cartilage and tracheal rings.

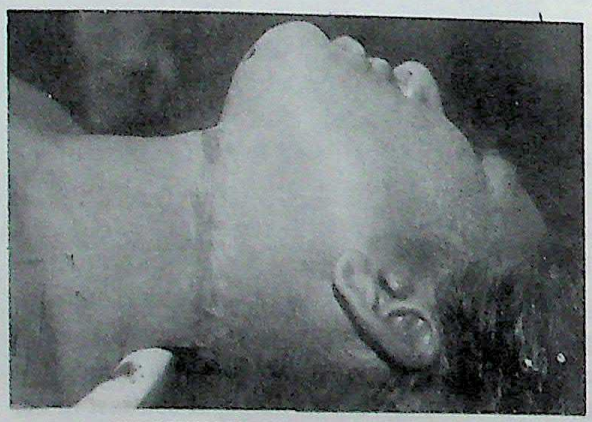

Fig 13.10 (a) Ligature mark of strangulation (front of neck) (b) Ligature mark of strangulation (back of neck)

Medico Legal Aspects

Strangulation is usually homicidal. Suicidal strangulation is rare, because once anoxia sets in, the pressure over the neck gets relaxed. In very rare cases, suicide may be effected successfully by strangulation when the victim manages to keep the ligature tight by knotting it, or twisting it, sometimes employing a stick as a lever. Accidental strangulation is rare, but it may occur when an article of clothing such as a dupatta, neck band, or cord is tightly drawn around the neck, in an abrupt manner.[11]

Box 13.2 details the infamous case of "**The Boston Strangler**", a serial rapist-murderer, who sexually assaulted and strangled 13 women in Boston, USA, in the 1960s.

It is important to differentiate between hanging and strangulation from the forensic point of view, if miscarriage of justice is to be avoided. Important differentiating points are mentioned in **Table 13.1**

Refer Table 13.1 for differences between Hanging and Strangulation

DROWNING

It is a form of asphyxial death which results from complete or partial submersion of a person in a fluid medium. Death occurs due to entry of fluid into the respiratory passages, or due to the effects of severe water and electrolyte imbalance.

MECHANISM

In order to understand the mechanism as well as the autopsy findings, it is necessary to appreciate what happens when a person (who cannot swim) falls into water. Initially, he sinks to a depth directly proportional to the momentum of the fall. The inherent buoyancy however tends to make the

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Table 13.1 Differences between Hanging and Strangulation

	HANGING	STRANGULATION
1. Ligature mark	Oblique, above the level of thyroid cartilage. Not encircling the neck completely.	Transverse, at or below the level of thyroid cartilage, completely encircling the neck with crossing of the ligature mark.
2 Injuries to neck structures	Under the ligature, on reflection of skin, tissues look pale. Bruising of neck muscles is less common.	Bruising of neck muscles is more common.
3. Hyoid bone	Fracture may occur	Fracture is uncommon
4. Thyroid cartilage	Fracture is less common.	Fracture is more common
5. Larynx and trachea	Fracture rare	Fracture may occur
6. Signs of asphyxia	Less prominent.	More prominent.
7. Bleeding	From ears, nose, and mouth, is less common	From external orifices is more common
8. Saliva	Dribbling often present	Absent

person float rather than sink. Additionally, his struggling movements, and the air trapped in the clothes help him to rise to the surface. In his desperate attempts at inhaling air, large gulps of water enter the throat, and may be partly inhaled and partly swallowed. The water which inadvertently enters the respiratory passages acts as a foreign irritant, and excites a strong reflex. Consequently, a large volume of air is driven out of the lungs. There is therefore a greater need for air, and the vicious cycle of drowning continues till a large quantity of water enters the lungs. Asphyxia consequently occurs, which leads to insensibility, and the victim finally succumbs. The barotrauma to the middle ear results in haemorrhages which causes vestibular disturbance, and this makes the victim lose his sense of direction under water. During the struggle for life under water, the victim may grasp anything within his reach. This accounts for objects such as weeds or mud occasionally found grasped in the hand of the victim, as a manifestation of cadaveric spasm.

TYPES OF DROWNING

Wet drowning

Water is inhaled into the lungs and the victim experiences severe chest pain.

Dry drowning

The water enters the larynx and is prevented from entering the lungs due to immediate and severe laryngeal spasm.

Secondary drowning (Post-immersion syndrome) Death occurs due to secondary effects of drowning, after a gap of half an hour to a few days following resuscitation. Death is due to cerebral

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anoxia and irreversible brain damage, associated with electrolyte disturbances and metabolic acidosis. Bronchopneumonia is also common.

Hydrocution (Immersion syndrome)

Death results from cardiac arrest due to vagal inhibition, as a result of cold-water stimulation of the nerve endings on the surface of the body, the epigastrium, and the ear and nasal passages.

Freshwater drowning

Drowning in fresh water causes dilution of the blood up to 70%, within a couple of minutes. This causes haemodilution resulting in lysis of RBCs, which in turn leads to hyperkalaemia and hyponatraemia. The circulation becomes overloaded. Fresh water also denatures surfactant, which causes decreased lung compliance. There is a fall in systolic blood pressure. Myocardial anoxia occurs, and death results from ventricular fibrillation.

Seawater drowning

Seawater constitutes a hypertonic medium. Due to haemoconcentration, as much as 40% of water from circulating blood is withdrawn into the lungs and massive pulmonary oedema is produced. This increases serum sodium concentration. Pulmonary oedema and myocardial anoxia cause death. Death in fresh water takes 4 -5 minutes, where as it is delayed upto 8-12 minutes in seawater.

CAUSES OF DEATH IN DROWNING

The causes of death in drowning could be asphyxia, ventricular fibrillation, myocardial anoxia, laryngeal spasm, vagal inhibition, exhaustion, or head injuries (from striking against objects during a fall into river, well, etc).

AUTOPSY FINDINGS

External Appearances

- The clothing is wet.
- The skin is wet, cold, and clammy. It appears pale due to contraction of superficial blood vessels.
- Body temperature falls to the temperature of the water in about 5 to 6 hours.
- Postmortem lividity is seen in the head, neck, and front of chest. It is usually bright pink in colour due to the imbibition of oxygen through water, but it may be dusky and cyanotic if the body had been floating face down, buttocks up, and hands and feet dangling down. If the water is turbulent and constantly overturns the body, postmortem staining may not be seen at all.
- *Cutis anserina* (goose fleshing), occurs due to contraction of erector pilorum muscles.
- Rigor mortis sets in and passes off early, due to exhaustion resulting from the person's struggle under water.
- Conjunctivae are often congested ; subconjunctival ecchymoses may be present.
- Hands affected by cadaveric spasm may contain various objects grasped while struggling in the water, such as seaweed, sand, straw, etc.
- After 48 to 72 hours or more, the skin of the palms and the soles becomes bleached, wrinkled, and soddened. This is called *washerwoman's hands and feet*. The epidermis separates from the dermis in glove and stocking fashion, from the hands and feet.
- Putrefaction leads to accumulation of gases, and the body rises to the surface of water to float (floater).

Froth

A fine, white, tenacious, persistent, frothy foam in

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the shape of a balloon, or mushroom, or horn, is invariably seen at the nose and mouth (Fig 13.11). This is formed by the water inhaled into the lungs, which acts as an irritant prompting the secretion of mucus. Water, air, desquammated epithelium, and mucus, are all churned up to form froth by violent respiratory efforts. Sometimes the froth is tinged pink due to blood from ruptured capillaries. This froth is pathognomonic of drowning. Copious froth exudes out when pressure is exerted on the chest.

Froth may also be seen in cases of poisoning due to opium, organophosphates, and snake bite, but it is never as copious as in drowning. However, in dry drowning, froth may not be present at all.

Fig 13.11 Typical froth in case of drowning

Internal Appearances

- Airways contain stiff foam or frothy fluid, or grit, gravel, mud, etc.
- Water in the air passages and stomach, especially dirty marshy water which is not potable, is an important finding to confirm death due to drowning.
- Lungs: Large subpleural hemorrhages may be produced due to rupture of interalveolar partitions beneath the pleura. These are called **Paltauff's haemorrhages**. They are more prominent over the lower lobes, and the interlobar surfaces. The lungs appear ballooned, oedematous, and have a heavy and boggy feel. They are doughy and spongy, water-logged, and pit on pressure. This is referred to as *emphysema aquosum*. The condition develops only when the conscious victim of drowning struggles for survival, whereas a related condition called *oedema aquosum* develops when a body is passively immersed in water, or when the victim is unconscious. It is a mere flooding of the lungs. Because of water-logging, rib marks are often seen on the lung surfaces. On cut section, watery, frothy fluid exudes out of the lungs. In deaths due to laryngeal spasm (**dry drowning**), these changes in the lungs and in the respiratory tree are not seen. In salt water drowning, the lungs appear purple, and are much more oedematous than in fresh water drowning. Further, in the latter case, lungs appear pinkish, and crepitus is heard on applying pressure.
- The blood is likely to remain in a fluid state, free from clots, due to prevention of coagulation resulting from the release of plasminogen activator from damaged endothelium of pulmonary capillaries.
- Water may be detected in the middle ear, resulting from violent respiratory excursions, which force the water into the ear. This never happens in *postmortem drowning*, i.e., submersion of the body after death, which is sometimes resorted to deliberately, for the purpose of misleading criminal investigation.
- Haemorrhages may occur in the middle ear and mastoid air cells, due to barotrauma produced by the pressure exerted by the surrounding water.

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DIATOM TEST

Most samples of water contain microscopic, unicellular algae or plankton called **diatoms**, which possess siliceous cell membranes, and vary in size from 10-80 microns. As a result of the hard cell wall, these micro-organisms are resistant to heat and acids. Diatoms get aspirated into the lungs along with inhaled water, and then pass into the venous circulation through ruptured pulmonary capillaries or alveolar walls. They are distributed to various organs of the body and the bone marrow. These diatoms are demonstrable histologically and their presence in the blood, viscera, or bone marrow along with their presence in control samples of water from where the body was fished out, is pathognomonic of drowning. A simple test can be performed on the sternal bone marrow to demonstrate these diatoms. The marrow is subjected to acid digestion, centrifuged, and the sediment examined under the microscope. A great advantage of this test is that diatoms resist putrefaction and are present even in highly decomposed bodies. A negative diatom test however does not rule out the possibility of drowning, since it could be a case of dry drowning.

The **Gettler test** (mentioned in some of the other Indian textbooks) is only of historical interest, and has no practical relevance.[12]

MEDICOLEGAL ASPECTS OF DROWNING

Most cases of drowning are either accidental or suicidal in nature. The former are more common in young children, elderly individuals, and non-swimmers, while the latter are more common in women. However in the USA, death due to drowning from any cause is said to be much more common in men than in women.[13] Homicidal cases are very rare. But cases do get reported from time to time.

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Box 13.1 The Case of Burke and Hare

In the early part of the 19th century in England, a hideous criminal trade flourished relating to the sale of dead bodies to medical schools. At that time, the laws were very strict regarding dissection of dead bodies. Anatomical study could only be done on corpses of executed criminals. As a result there was an acute and perpetual shortage of cadavers. It was to plug this deficiency that a special breed of individuals emerged who came to be known as "Resurrectionists." They had a simple (though macabre) solution : dig out freshly buried bodies surreptitiously from cemeteries or graveyards and supply them to eager anatomists for a hefty price! Naturally the trade by its very nature, could be accomplished only through clandestine nocturnal expeditions involving a trip to the grave, a quick exhumation, the stripping of valuables and belongings from the corpse, and the delivery of the quite-naked body to the hospital. Some of the resurrectionists became experts at the trade, and could sometimes complete the whole process in 20 minutes, counting from the moment when they climbed over the cemetery wall down to the moment of delivery of the body. Much like an efficient baker delivering fresh bread in a jiffy straight from the oven!

The evil fame of the resurrectionists spread all across England, and the inhabitants found it necessary to band themselves together in order to protect their cemeteries at night. The citizens would mount guard in turns with loaded guns.

The Edinburgh Medical School was particularly famous for procuring bodies regularly through resurrectionists. The Chief Anatomist (and Surgeon), Dr. Knox was never at a loss for cadavers and kept his hospital well stocked at all times. His chief suppliers were an ingenious duo named William Burke and William Hare. They had realised early on in their 'professional' career that if they depended only on cemeteries for procuring dead bodies, their trade would flounder. After all, people did not die every day. So they struck upon a gruesome plan of 'generating' dead bodies in order to maintain a regular supply. They actually hit upon the idea by chance, when an old soldier who owed them some money died suddenly in their lodgings. They transported the body to Dr. Knox who promptly received it and paid them a handsome price (since the body was in a neat and fresh condition). Encouraged by this, Burke and Hare decided to kill people regularly, thereby doing away with the element of chance altogether. The ugly process of exhumation could also be avoided. Over a period of time, Burke perfected a method which they employed with great success. The modus operandi consisted of roaming the disreputable parts of the city at night and picking up tramps,

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beggars, or derelicts. They would invite the poor homeless victim to their living quarters with promises of drink, food, and merriment. There, they would drink together heavily, and Burke, who possessed a fine voice would begin to sing. As soon as their guest was drunk enough, Hare would wrestle him to the floor and close his mouth and nostrils with his hands, while Burke would sit on the poor victim's chest. Thus an effective combination of intoxication and asphyxia (involving smothering and traumatic asphyxia), would result in a quick, non-messy death, and the blemishless body would then be handed over to Dr. Knox who never asked awkward questions.

In this manner, Burke and Hare succeeded sixteen times without arousing suspicion, but on the seventeenth instance, a suspicious neighbour informed the police of "curious goings-on" next door. The police searched the lodgings of the macabre duo and discovered the body of an old woman in a box, ready for supply to Dr. Knox. Both Burke and Hare were arrested. The latter turned approver and confessed everything. Burke was hanged publicly on a scaffold erected in an open space called Lawnmarket, in Edinburgh, on 28 January 1829. His confederate William Hare, as well as the one-eyed surgeon Dr. Knox went scot-free. To this day, a small song sung by the people who had gathered to see Burke's public execution, remains popular -

"Burke's the murderer
Hare's the thief,
And Knox the old boy
Who buys the beef !"

Box 13.2 The Boston Strangler

Between June 1962 and January 1964, thirteen women were sexually assaulted and then strangled to death in the Boston area of Massachusetts, USA, by a fiend whom the press dubbed the "Boston Strangler." This man proved to be Albert Henry DeSalvo. Born in 1931, DeSalvo was one of six children. He began stealing when he was in his teens, and was charged with breaking and entering numerous times before joining the army at age seventeen. He served in the occupation forces in Germany, where he began boxing on a U.S. army team. Small, but squat and tough, DeSalvo became the U.S. Army welterweight and, while stationed in Frankfurt, met and married a petite German girl.

Returning to the U.S. with his new wife, DeSalvo was stationed at Fort Dix, New Jersey, where in January 1955, he was charged with molesting a 9-year-old girl, his first sex offense. The child's mother, fearing publicity would affect her daughter, refused to prosecute. The Army, therefore, had no case against DeSalvo and released him. He received an honorable discharge a short time later and moved back to Boston with his wife. DeSalvo worked as a handyman, and he and his wife had two small children.

DeSalvo's sex drive was almost overwhelming. He exhausted his wife Irmgard, who told him to control himself. DeSalvo demanded sex from her on an almost non-stop basis. He thought about sex night and day, according to his later admissions, and he found little release. He was arrested for breaking and entering and given a suspended sentence in 1958, and a short time later he embarked on a sexual game whereby he became known to the police as "Measuring Man". DeSalvo approached young, attractive women in their apartments, telling them he represented a modelling agency and

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that they had been selected as possible candidates for modelling in television commercials. If chosen, they could make considerable money and would become famous, and they might even be offered a movie contract. Hundreds of young women opened their apartment doors to him after hearing this pitch and DeSalvo, clipboard and tape measure in hand, would then measure the woman's vital statistics. At these times DeSalvo would not make overt sexual approaches, but he did seduce a number of these gullible females.

On Mar. 17, 1960, police responded to an alarm of a break-in and they chased and caught DeSalvo. He had thrown away a screwdriver which he had been using to force apartment door locks, but this was recovered. On his person was found a pair of gloves and a tailor's measuring tape. He admitted that he was the "Measuring Man" about whom police had received several complaints. DeSalvo received a two-year sentence for breaking and entering, and was released in ten months. When DeSalvo was released from prison, he returned to his family, but now became more aggressive, breaking into apartments and tying up and raping females. He was described by his victims as the "Green Man", since he wore green workpants and shirt.

In the summer of 1962, DeSalvo began to add murder to his sexual attacks, raping and strangling his victims, the first of whom was 55-year-old Anna Slesers, found in her apartment on Gainsborough Street in Boston, her body placed in a lascivious position. DeSalvo had used a cord to strangle his victim and had tied the ends in a bow beneath her chin, a technique he would continue to employ, as if it were his trademark. Within two weeks, DeSalvo attacked and killed an 85 year-old woman, Mary Mullen, a victim he later reluctantly talked about since she reminded him of his grandmother. On June 30, 1962, DeSalvo raped and strangled Helen Blake, a 65-year-old nurse. Next was Nina Nichols, a woman in her sixties. She fought her attacker, digging her nails into his arms as he strangled her from behind.

On Aug. 19, DeSalvo raped and strangled 75-year-old Ida Irga. Jane Sullivan, sixty-seven, died at DeSalvo's hands the next day. Boston police were inundated with demands to solve the rash of horrible rape-murders. Though scores of sexual deviants were arrested and questioned, DeSalvo escaped attention. DeSalvo refrained from making more attacks until Dec.5, 1962, his wedding anniversary. He spotted an attractive girl entering an apartment and followed her. He knocked on her door and employed the usual technique that invariably gained his access to an apartment, saying through the door that he was a repairman sent by the landlord to check the pipes and toilet. The girl, however, refused to let him inside. He went to another apartment and knocked on the door. Inside was 25-year-old Sophie Clark, a tall, attractive black woman. DeSalvo persuaded her to open the door, saying that he was from a modelling company, using his old Measuring Man technique. As he walked inside the apartment, Clark turned her back on DeSalvo, who later reported that he was stunned by her curvaceous body. He leaped on her from behind, subduing her, raping her and strangling her, leaving her as he had the others, her naked body propped upward, legs spread, the bow tied beneath the cord he had used to strangle her.

His next victim was Patricia Bissette, a 21-year-old secretary he had visited years earlier as Measuring Man. On Dec 8, 1962, she invited DeSalvo inside, gave him a cup of coffee, and when she turned her back, he placed his arm around her throat and raped her, strangling her with her own nylons. On Mar 9, 1963, 69-year-old Mary Brown allowed DeSalvo into her apartment, thinking him a workman sent by the landlord to fix her stove. This time DeSalvo's violence was unchecked. He had brought along a lead pipe, which he used to crush his victim's head. He raped Brown *after* killing her. He then drove a fork into her breast several times, leaving it embedded in the flesh. He also strangled her, although by that time, she was dead. On May 6, 1963, instead of driving to work,

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DeSalvo drove to Cambridge, "on an impulse" he later said, and there spotted pretty Beverly Samans, a 23-year-old undergraduate living on University Road. He gained entrance to her apartment, tying her to bedposts. He then blindfolded and gagged her and then repeatedly raped her. He used the girl's nylon stockings to strangle her. Before leaving his victim, DeSalvo got out his jackknife and began stabbing the girl. The autopsy reported twenty-two stab wounds.

DeSalvo's eleventh victim was 58-year-old Evelyn Corbin, whom he strangled and raped on Sept 8, 1963. When Corbin failed to make an appointment, police were summoned and they found the woman as they had the others, except that she had been manually strangled. The killer had left his trademark, a nylon with a bow, tied about her ankle. Boston police seemed helpless to catch the killer. The city was in a near panic with thousands of husbands constantly calling their wives or staying home from work to protect them against a fiend who seemed to come and go at will. Not a single person reported seeing this mass killer and police were admittedly stymied.

A special "Strangler Bureau" was set up and a dedicated detective force began running down the slimmest clues around the clock. Dozens of sex offenders, muggers, and even peeping toms were rounded up, questioned, and subsequently released. On Nov 23, 1963, a day following President John F. Kennedy's assassination in Dallas, Texas, DeSalvo struck again gaining entrance to the apartment of Joann Graff, a 23-year-old dress designer. He raped and strangled her with her own black leotards, tying these in a bow about her neck.

On Jan 4, 1964, DeSalvo struck for the last time, claiming his thirteenth victim, 19-year-old Mary Sullivan. Once inside her apartment, he flourished a knife, tied up the girl and raped her. He then strangled her with his hands. He left her naked, and as a bizarre afterthought, inserted a broom into her and placed a card he found in the apartment between her toes which read, "Happy New Year". In Fall 1964, a young woman reported being sexually assaulted in her apartment, describing a man police identified as DeSalvo, using the same technique as he had when labelled the Measuring Man. DeSalvo was arrested for breaking and entering, held on a $100,000 bond, and sent to the mental institution at Bridgewater. Amazingly, police never coupled DeSalvo with the crimes of the Boston Strangler.

At Bridgewater, DeSalvo claimed that he was hearing voices and he was diagnosed by psychiatrists as "schizophrenic". On Feb 4, 1965, he was ordered to be detained indeterminately. Another inmate at Bridgewater, George Nassar, who had killed a garage attendant and who was himself a suspect in the Strangler killings, met DeSalvo and after listening to him talk for some time, mostly about sex and violence, came to believe DeSalvo was the Boston Strangler. He informed his lawyer, who interviewed DeSalvo, recording his conversations. DeSalvo admitted being the Strangler and even added two killings to the known murder count of thirteen. More importantly, he related facts about the murders which the police had kept secret, the positioning of the naked bodies, the tying of bows in the strangling cords, nylons, and pantyhose, the wounds inflicted, all the sordid details that only the killer would have known.

Police were still puzzled since they had no eyewitnesses who could positively identify DeSalvo. They were disinclined to officially prosecute him as a mental patient. He was kept in confinement and the strangulation murders stopped. Later, DeSalvo was transferred to the Walpole State Prison, where in his cell, on Nov 26, 1973, he was found dead, stabbed to death through his heart.

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CHAPTER - 14

Surgical and Anaesthetic Deaths

"Man can but achieve transitory sleep, while the Good Lord can put him to eternal sleep".

- Dr. Graham Pearce

Anaesthesia, the panacea for pain has improved the quality of all surgical procedures, since management of pain had been the prime concern in such procedures. However, it is also true that the agents which relieve pain are themselves often the source of morbidity and mortality, for any intervention (surgical or anaesthetic), does carry with it an element of risk to the life of the patient.

Many investigators have studied the role of anaesthetic mortality in various surgical procedures. Deaths occuring within 24 hours, 48 hours, and 6 post-operative days, and in patients failing to regain consciousness following anaesthesia, have been included in these studies. According to the 1987 Report of the Confidential Enquiry into Perioperative deaths, of the 4000 cases studied, anaesthesia was considered to have been the sole cause of death in 0.1%, and a contributory factor in some 14% of deaths.[1]

Fatalities associated with surgical intervention or invasive diagnostic procedures can be separated into several categories[2] :

1. *Those directly caused by the disease or injury for which the operation or anaesthetic was necessitated -*
   In the advanced stage of some diseases, a heroic effort may have to be taken to save the life of the patient, and if such intervention leads to the death of the patient, the disease from which he was suffering is cited as the cause of death, and the intervening procedure is not taken into account. But in cases where the patient had been seen walking into the operation theatre and is subsequently brought out dead, the suspicion of the relatives may be aroused, and such death might lead to an investigation.

The American Society of Anesthesiologists have classified deaths during surgical procedures as follows:

- ASA RISK I: Normal healthy patient.
- ASA RISK II: Mild systemic disease.
- ASA RISK III: Severe systemic disease limiting activity but not incapacitating.
- ASA RISK IV: Incapacitating systemic disease that is a constant threat to life.
- ASA RISK V: Moribund patient not expected to survive 24h without an operation.
- E: Emergency operation.

Categories I, II and III require full medicolegal investigation, as death is unexpected in these cases.

It is important to note that in those cases where surgical operation was done in a victim of

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trauma, it may be impossible to separate the relative contribution of each to death.

2. *Those caused by a disease or abnormality other than that for which the procedure was being carried out* -
In case death was caused by a disease other than that for which the procedure was carried out, and its existence was known beforehand, the pathologist must evaluate the need for the operation. However, if the disease had not been suspected by the clinicians, or if specialised diagnostic procedures were not available, the death can be excused. But the presence of common conditions such as hypertention, ischaemic heart disease, lung disease, etc., can lead to medico-legal problems, if proper care had not been taken in their diagnosis or treatment.

3. *Surgical or diagnostic procedure mishap* -
Failure of a surgical technique might be inadvertent as happens in some unusually difficult operative circumstances, or anatomical abnormalities, or it may be accidental due to failure of the equipment, or it might be a result of error or incompetence on the part of the surgeon, in which case he can be sued for negligence.

4. *Mishap or complication of anaesthetic administration* -
a) Overdosage of anaesthetic agent:
True anaesthetic deaths arising out of overdosage of the anaesthetic agent are very rare, and if at all they do occur, it is due to ill-advised polypharmacy.[3] Though data regarding the blood levels of various anaesthetic agents at different grades of anaesthesia are available in the literature, they are of limited use in practice. The current trend is to achieve a light plane of unconsciousness with a barbiturate or some other hypnotic, accompanied by the inhalation of nitrous oxide and oxygen, along with small quantities of a liquid agent like halothane. Muscular relaxation is achieved by the use of relaxants. Anaesthetic gases are known to cause dependence (physical or psychological), among anesthesiologists, dentists, and paramedical personnel to whom they are readily available. *Etheromania* is the dependence caused by ether, and is characterised by rapid onset and sudden abatement. Etheromania is of two types - acute and subacute. In the acute variety, the person becomes delirious, and suffers from hypomania and convulsions leading to death. In the subacute type, the person becomes aggressive and psychotic. It is usually difficult to determine whether the death was due to accidental overdosage or due to suicidal ingestion in such cases.

Muscular relaxation is achieved by the use of relaxants such as gallamine, pancuronium bromide, tubocurarine, etc., which compete with acetylcholine at the motor end plate, or with agents such as succinylcholine, decamethonium iodide, etc., which depolarize the motor end plate rendering it inexcitable to acetylcholine. Whatever be the mode of action, the end result is the same - paralysis, the only difference being the former are long acting agents, while the latter are short acting. Overdose with tubocurarine and pancuronium bromide can be treated with neostigmine, while prolonged artificial respiration is the only solution to overdosage with succinyl choline and similar agents.

b) Anaesthetic misadventure:
"An anaesthetic misadventure or a *critical incident* as it is called, is a human error or equipment failure that could have led (if not discovered or corrected in time), or did lead to an undesirable outcome, ranging from increased length of hospital stay to death."[4]

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Most cases of anaesthetic morbidity and mortality occur from one of the following causes –

1. Complications of anaesthetic procedure
2. Human error
3. Equipment failure

Some of the **complications** arising out of **anaesthetic procedures** (either due to inherent risks of the procedure, human error, or equipment failure), are listed in **Table 14.1.**

A variety of factors associated with **human error** have been reported : emergency case, lack of experience with technique or equipment, failure to perform a normal check, lack of skilled assistance, restricted access to patient, and inadequate supervision of the patient due to distraction, fatigue, inattention, boredom, or anxiety.[5]

Cooper, Newbower and **Kitz** have described three categories of human error.[4] :

a) *Technical* - in which the action taken is not the action intended, arising from deficiencies of technical skill, or from poor design of the equipment or apparatus.
b) *Judgemental* - in which the action represents a bad decision, arising from lapses in training or poorly developed decision making skills.
c) *Monitoring & vigilance failures* - in which the essence is a failure to recognise or act upon visible data requiring a response.

The following preventive strategies have been suggested:

1. **Training and Supervision** - Observation by the authors indicated that some critical incidents were associated with inexperience or inadequate training. Inadequate supervision by seniors also has been cited as a cause. Inadequate knowledge about a new device/technique also leads to error. All these can be rectified by regular in-house training of the anaesthetists.

Refer Table 14.1 for Complications of Anaesthetic Procedures .

2. **Specific Protocol Development, More Complete Preoperative Assessment, and Equipment and Apparatus Inspection** -
Pre-operative assessment of patients, and preoperative inspection of equipment and apparatus will prevent a number of critical incidents. A standard checklist procedure similar to the one used by pilots and co-pilots in aviation is suggested to inspect the equipments, which can vary from one institution to the other according to the nature and availability of the instruments.[6]

3. **Additional Monitoring Instrumentation and Equipment, and Human Factors Improvements** - Improved standardization or arrangements of drugs in the anaesthesia workspace, and improvements in design of gas-flow control knobs and breathing circuit scavenging connections are now mandated by the national standards for new apparatus.

The standards recommended by the American Society of Anaesthesiologists for basic patient monitoring include arterial blood pressure recorder electrocardiography, an oxygen analyzer, and a ventilator disconnection alarm. Monitors that are encouraged but not mandatory, include pulse oximetry, capnography, and spirometry. Most monitors are fitted with alarm systems, usually with preset and modifiable threshholds, which produce an auditory signal when a high or low limit is passed.[7] A survey by **Kestin, Miller** and **Lockhardt** on the significance of auditory alarms that sound during routine anaesthetic management, revealed that 75% of all alarms that sounded were spurious alarms, while only 3% indicated risk to the patient. 99% of all alarms were from pulse oximeter, ECG or BP monitors, the common sources of interference

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Table 14.1 Complications of anaesthetic procedures

Complication of / Due to	Etiology	Outcome	Management
1. Upper airway obstruction	Anaesthetic agents cause an alteration in the pattern of activation of muscles which maintain the upper airway.	Hypoxia leads to a. impairment of gas exchange b. regurgitation & aspiration of gastric contents	Premedication with anticholinergics to reduce secretions, topical analgesia of the airway, and muscle relaxants
2. Laryngo-spasm	Performing laryngoscopy and surgery under light anaesthesia, presence of foreign matter such as blood, saliva, sputum or vomitus on or near the vocal cords. Inhalation of irritant vapours.	c. pulmonary oedema d. cardiovascular disturbance	
3. Difficult laryngoscopy	Conditions which cause difficulty in opening the mouth, or extending the head at occipito-atlanto-axial complex. Conditions causing airway obstruction variants of normal small mouth:(large tongue, receding jaw); acromegaly, thyroid enlargement, bulky tonsils, etc.	Upper airway obstruction and laryngospasm, leading to hypoxia	Pre-operative assessment prior to anaesthesia regarding head extension, mouth size, dentition etc. X-ray to detect upper airway obstruction. Use of antisialogogues i.m. preoperatively
4. Laryngoscopy and tracheal intubation		a. Gingival damage b. Lingual nerve damage c. Cardiovascular responses d. Increased intraocular pressure	Proper assessment and treatment of cardiovascular diseases, and proper technique of anaesthesia based on intravenous opioid analgesics. Suxamethonium is contraindication
5. Endotracheal intubation	Use of excessive force Nasal intubation	Perforation of pyriform fossa/ posterior pharyngeal mucosa/cervical oesophagus. Trauma to nasal passages. Oesophageal intubation	Gentle insertion, well lubricated tube, and use of a. Oesophageal detector device b. Capnographhy c. Direct visualisation of tracheal rings with fibre optic laryngoscope.

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6. Dental damage	Insertion and removal of laryngoscope, fragile dentition		Use of a tooth shield. Use of gumelastic, or neoprene oesophageal bougie
7. Ocular damage	Surgical drapes, instruments, hands, anaesthetic hardware, facemasks	Corneal abrasions, retinal detachments and retinal artery thrombosis	Closure of eyes with a tape Appropriate size of face mask with liberal lubrication
8. Pharyngeal airway	Long airways irritate the larynx	Laryngospasm	Use of correct length and diameter
9. Laryngeal mask airway		Laryngospasm, as the effect of muscle relaxant wanes at the end	Use of capnograph and airway pressure monitor to detect obstruction
10. Throat pack	Failure to remove it prior to extubation	Complex airway obstruction	
11. Harnesses	Tight straps	Facial nerve palsy, and blindness due to retinal artery thrombosis	
12. Fire in the airway	Endotracheal tube ignition in laser surgery with CO_2 laser beams, especially with high concentrations of oxygen or nitrous oxide		Non combustible tube. Wrapping the endotracheal tube with reflective tape. Moist ribbon gauze placed around the tube above the cuff. Cuff filled with physiological saline
13. Regional, epidural, and spinal anaesthesia	Puncture of and injection into the spinal cord and nerve roots	a. Haematoma b. Paresthesia c. Anaesthetic effect in adjacent structures leading to cardiac arrest d. Systemic toxicity of local anesthesia e. Myelopathy f. Arachnoiditis	
14. General anaesthesia		Hepatic and renal complications. Halothane hepatitis.	
15. Malignant hyperthermia	Autosomal dominant inherited trait, plus succinylcholine and halogenated anaesthetic agents	Sudden rise in temperature, stiffening of skeletal muscles, rapid heart rate, and respiratory distress	Diagnosis by elevated creatine phosphokinase etc.
16. Supplementary oxygen	Inlet port of the oxygen mask loosely applied over the open end of tracheal tube, with/without the cuff inflated.		

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being from electrocautery or operating room lights. Various suggestions to improve the alarm system have been put forth, such as visual as well as auditory indication, inability to permanently silence the alarm, new alarm sounds similar to a segment of a bird song, repeated alarms with a period of silence in between, etc.

4. **Organizational Improvements -**
The deployment of properly trained personnel will alleviate most of the critical incidents. Human errors were associated frequently with lack of sleep and fatigue. Regulation of working hours of anaesthetists/medical staff will minimise the problem.[8]

Equipment Failure

Equipment failure is a relatively minor cause of anaesthetic mishaps. **Cooper, Newbower** and **Kitz** reported that equipment failure accounted for only 11% of all critical incidents, and that only 4% of the incidents with substantive negative outcomes involved equipment failure.[5]

MEDICOLEGAL PROBLEMS[9]

Failure of empathy and communication towards the patient and his relatives are the main precipitating factors of negligence suits. Anaesthetists and surgeons should ensure the following in order to minimise the possibility of malpractice actions:

1. Always establish a proper physician-patient relationship.
2. Establishment of identity is important in surgery, which also includes the type of surgery to be performed, the side (in bilateral structures), self-identification, i.e., the patient must feel the presence of the doctor during surgery, and lastly re-identification, so that the patient sees the familiar face of doctor on awakening.
3. Informed consent of the patient (if not possible, that of a relative) with particular regard to the physical status of the patient, and associated disabilities must be obtained. Legally, separate consent for anaesthesia apart from surgery has to be taken.
4. Documentation regarding pre-anaesthetic evaluation and assessment of the patient and pre-operative record of events, is of vital importance. *Automated anaesthesia records systems* similar to the 'black-box' in aviation are available, which are clearer and more complete than handwritten records, reduce the workload of the anaesthetist, and can prove to be a valuable tool for retrospective analysis of anaesthetic mishaps.[10]
5. Regarding any new instrumentation/technology, it is imperative to ascertain whether the anaesthetist had acquired the necessary skill to operate it, by formal training or by the demonstration of a salesperson, as also the rate of success of the procedure in his hands.

AUTOPSY PROCEDURE[2]

1. Full clinical information is essential, especially in so called 'anaesthetic deaths', since the morphological findings may be minimal or even absent.
2. Surgical intervention and its sequelae such as exudate, sepsis, haemorrhage, oedema etc., may make the dissection difficult, especially if the particulars of the surgical procedure are not fully known. Postmortem changes can further complicate the appearances. Resuscitative intubation can cause artifactual injury that may mimic inflicted injuries caused by neck compression, including manual strangulation and law enforcement "neck holds."
3. Surgical and anaesthetic devices introduced into the patient, such as airways, endotracheal tubes, indwelling needles, catheters, monitoring electrodes, etc., should not be removed prior to autopsy.

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4. Position of the endotracheal tube must be checked by a pre-autopsy radiograph. A ring of oedematous oesophageal mucosa at the level of the tube in the trachea, and distension of stomach and intestines indicate oesophageal intubation.[11]
5. Ante-mortem blood or body-fluid samples if available, should be retained for analytical tests.
6. The presence of the surgeon and the anaesthetist concerned, at autopsy, will make the interpretation of the findings easier.
7. Care must be taken to detect any surgical emphysema, pneumothorax, or air embolism.

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CHAPTER - 15

Starvation

"The old can withstand starvation better than the young".

- Hippocrates

Starvation results from the deprivation of a regular and constant supply of food, which is necessary for the maintenance of the nutritional balance of the body. The term *starvation* includes both *subnutrition* (intake of an insufficient quantity of food), as well as *malnutrition* (feeding of inadequate quality). Malnutrition can and does occur as a separate entity, while subnutrition must always include some degree of malnutrition.[1] Starvation is regarded as **acute**, when the necessary quantity of food and water are suddenly and completely withheld, and **chronic**, when there is a gradual deficiency in the supply of food. In India, starvation deaths due to famine is not an unknown occurrence, though it is not so common today. *Inanition* is the opposite of obesity, while *cachexia* simply means severe inanition.[2]

Neglect and starvation are not synonymous, but are often so closely associated, that the two conditions are usually considered together. Death can occur from deliberate or negligent lack of provision of proper care and nutrition of children. In general, though there are many exceptions, nutritional neglect and physical battering tend to be mutually exclusive. The abused child is usually well nourished, while the emaciated child is usually uninjured.[3]

GENERAL FEATURES

A minimum of 1900 calories of food per day is the average dietary requirement for an adult, below which the effects of starvation will begin to manifest. There is danger to life when more than 40 percent of the original body weight has been lost, though the speed of loss is relevant. Total lack of food is likely to cause death in about 50-60 days if adequate water is available, while if it is not, death is greatly accelerated. Total deprivation of water results in death in about 10 days. The rapidity of death also depends on the weather, temperature of the environment, and the original fitness (or fatness!) of the individual.

Signs and symptoms of starvation

- Begin with an acute feeling of hunger for the first 2 days.
- Loss of fat and emaciation make their appearance in about 4 days.
- There is progressive loss of body weight.
- The skin becomes dry, pigmented, shrivelled, thin, parchment-like, fissured, and drawn tight over bony prominences.
- The cheeks become sunken, and lips are dry and cracked.
- The abdomen becomes scaphoid.
- The person feels extremely weak, and may look pale and cadaverous. Pallor is due to anaemia of nutritional type, resulting from inadequate intake of iron, proteins, vitamins and other required elements.

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- Pupils get dilated.
- Tongue is dry and coated. The breath turns offensive.
- Hair becomes dry, lustreless, and brittle.
- Nails become brittle and ridged.
- Pulse is feeble, and blood pressure is low because of cardiac atrophy.
- There may be oedema of the feet due to decrease in blood proteins (hypoproteinemic oedema).
- The temperature is subnormal.
- There is constipation at first, but later diarrhoea sets in.
- Urine is scanty and dark coloured.
- The mind usually remains clear till the end. Sometimes the person becomes delirious just before death.

FACTORS MODIFYING THE EFFECTS OF STARVATION

a) **Age**: Old people generally withstand the effects of starvation better than young adults because of their lower food requirements. Similarly, a mature adult can withstand starvation better than a growing child.
b) **Sex**: Women can usually combat starvation better than men for the same reason as above, as also the fact that they generally possess more body fat.
c) **State of health**: Healthy people can endure the effects of starvation better than the sick.
d) **Body fat**: Obese people can withstand the effects of starvation better than the lean.
e) **Cold** : Exposure to cold aggravates the effects of starvation, because it accelerates the basal metabolic rate.

TREATMENT

Rest and warmth must be provided and maintained. Solid food must not be given straight away. Small sips of warm water mixed with glucose or fruit juice can be given at frequent intervals. Later solid food can be given in gradually increasing quantities. Administration of protein hydrolysate helps to raise the level of blood proteins and thus combat oedema.

POSTMORTEM FINDINGS

External

1. Emaciation : The degree of loss of weight is an indication of the duration of starvation.
2. Pupils : dilated.
3. Skin : dry, shrivelled, thin, parchment-like, fissured, and drawn tight over bony prominences. Trophic ulcers may be present.
4. Hair : dry, lustreless, and brittle.
5. Nails : brittle and ridged.
6: Lips : dry and cracked.
7. Tongue : dry and coated.
8. There may be oedema of the feet.

Internal

1. Loss of body fat and atrophy of muscles.
2. Stomach and small intestine: small and contracted.
3. Gall bladder : distended with bile.
4. Omentum and mesentery : devoid of fat.
5. Large intestine : contains hard faecal matter.
6. All organs except brain, shrunken and reduced in size. Brain is often oedematous.
7. Bones: dimineralised.

CAUSE OF DEATH

In acute starvation, death occurs from inanition and circulatory failure. In partial/chronic starvation, the vitality of the individual is reduced, and death may result from intercurrent disease.[4]

MEDICO-LEGAL IMPORTANCE

Starvation may be accidental, suicidal, or homicidal. In homicidal cases, the victim is usually an infant who has been deliberately and wilfully deprived of food. Successful prosecution in cases of fatal

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infant starvation depends on establishing the fact that the death resulted from failure to offer the child sufficient food. This deficiency must be coupled with evidence that the mother was aware of the gravity of the child's condition, and that she did nothing to ameliorate the situation in spite of availability of resources.

The most horrific example of mass homicide by starvation was perpetrated by Nazi Germans on helpless Jews during the second world war. Many deaths from starvation took place in Nazi concentration camps in Belsen and other places in Germany during that time. At Belsen, which was perhaps the worst camp of all, the entire organisation for the provision and distribution of food broke down during the last fortnight prior to liberation, and only those capable of fighting for food, managed to obtain the paltry supply available, which was just half a litre of soup per day ![5]

Suicidal starvation is rare, though it may be seen among lunatics, and occasionally among prisoners who go on a hunger strike as a form of protest. Voluntary starvation for religious or political reasons is well known.

In cases of accidental starvation, the circumstances are self-explanatory, such as famine, ship-wreck, entombment in mines, or diseases such as stricture of the oesophagus.

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CHAPTER - 16

Impotence, Frigidity and Sterility

"If his bones in general, and his shoulders in particular are strongly made,
If his gait and voice are vigorous
By these tokens may a potent man be known
And one impotent by the opposite characteristics".

- Narada Smrithi

IMPOTENCE

Impotence, in its simplest meaning, is the incapability to perform the act of coitus. Since in this act the male is usually the active partner and the female is relatively passive, the term **impotence** is generally applied to *the inability of the male to achieve and/or maintain penile erection and thus engage in copulation.*[1] Further, since erection involves complex psychological, neurological, hormonal, arterial, venous, and sinusoidal factors, and because the prime purpose of sexual intercourse is procreation, **impotence** can be more completely defined as *male sexual dysfunction involving inadequacy of erection or problems with emission, ejaculation or orgasm,*[2] and on a psychological basis as *persistent or recurrent partial or complete failure in a male to attain or maintain erection until completion of sexual activity.*[3] Impotence may be either temporary or permanent.

FRIGIDITY

Frigidity or sexual unresponsiveness, is the analogue of impotence in the female. In other words, it refers to a difficulty or inability to take part in sexual intercourse by a female. Like impotence, frigidity could be either temporary or permanent.

CAUSES OF IMPOTENCE

Apart from the fact that physiological impotence occurs at the extremes of age, the causes of impotence may be organic, atonic, paretic or functional (psychological).

These will be considered below in some detail.

1. **Organic Causes of Impotence** are due to structural abnormalities in the reproductive system, and may be either *congenital* or *acquired.*
   a. *Congenital causes* include Klinefelter's syndrome, primary testicular failure, cryptorchidism, and congenital deformities (e.g., phimosis, epispadias, accessory penis and bent-nail syndrome).
   b. *Acquired causes* include partial/total amputation of the penis, pre-pubertal castration, and local diseases (e.g., inflammatory hydrocoele, filariasis scroti, infections such as gonorrhoea, and carcinomata).

2. **Atonic Causes** are due to paralysis of the motor nerves supplying the genitalia without evidence of any lesion of the central nervous system, and may be either

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a. *traumatic*, such as a blow to the external genitalia or injury to the cauda equina,
b. *pharmacogenic*, because of excessive doses or prolonged use/abuse of sedatives, parasymphatholytic agents, ethyl alcohol, opium, or cocaine, or
c. because of *diseases* such as autonomic neuropathies of varying aetiology or tumours of the cauda equina.

3. **Paretic Causes** are due to lesions in the central nervous system or spinal cord, including
   a. hemiparesis or paraplegia, as a result of either trauma or a cerebrovascular accident,
   b. general paralysis of the insane, or
   c. drugs.

4. **Functional Causes** are dependent upon the mental complex, and are usually due to
   a. fear, timidity and anxiety, all of which are responsible for what is commonly described as *first night* or *bridegroom impotence*,
   b. aversion to a particular female (*impotentia quoad persona*) or to females in general (*sexual aversion disorder*, defined as a "persistent or recurrent aversion to, and avoidance of, all or almost all, genital sexual contact with a sexual partner"[3])
   c. excessive passion (leading to premature ejaculation), or
   d. sexual overindulgence.

MEDICOLEGAL IMPORTANCE OF IMPOTENCE

Civil Cases

- Legally, a marriage is a contract between man and wife, in which sexual union between the two is presupposed. If there is any cause, in either partner, which prevents such sexual intercourse (such as impotence existing either before or at the time of the marriage), the marriage cannot be consummated, and can therefore be declared by a court of law to be *null and void*. However, when impotence develops after the marriage has been consummated, it can only be cited as a ground for *divorce*, whether or not the consummation has resulted in conception and the production of offspring.
- Similarly, in a case of *disputed paternity*, the putative father may claim that he is impotent, and therefore could not have fathered the child.
- A man may claim *compensation* for an injury that has allegedly rendered him impotent.

Criminal Cases

In criminal cases of alleged *natural* or *unnatural sexual offences*, the accused may plead that he is impotent, and so cannot be charged with the crime.

The role of a registered medical practitioner in the cases listed above is obvious. However, in no instance can he positively affirm that a person is potent. Rather, should he, on examination, find that a male is physically normal in all respects, including development of secondary sexual characteristics and external genitalia, and if all the causes of impotence enumerated above are excluded, he is justified in opining that he "finds nothing to suggest that the person is incapable of participating in normal sexual intercourse." In other words, a **medical certificate of potency** should be couched in **negative language**.

CAUSES OF FRIGIDITY

1. **Permanent Frigidity** is invariably *psychogenic*, resulting most often from either sexual abuse during childhood, or a traumatic sexual assault during adulthood, manifesting as either a *sexual aversion disorder* (see **impotence**) or as a *female sexual arousal disorder* ("persistent or recurrent partial or complete failure to attain or maintain the lubrication-swelling response of sexual excitement until completion of the sexual

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activity, OR persistent or recurrent lack of a subjective sense of sexual excitement and pleasure in a female during sexual activity"[3]).

2. **Temporary Frigidity** manifests only as *female sexual arousal disorder*. It may be due to either
   a. **dyspareunia**, a term loosely used to describe both difficult and painful coitus, or
   b. **vaginismus**, which is hyperaesthesia leading to
      i. painful spasms of the sphincter vaginae and levator ani, with
      ii. simultaneous spasmodic contractions of the adductor muscles of the thighs and the erector spinae,
      iii. making penetration impossible.

Vaginismus may be either *primary* (where there is no organic lesion), *secondary* (due to a painful local inflammation or lesion, or during menstruation), or *psychogenic* (due to fear, timidity, or anxiety).

MEDICOLEGAL ASPECTS OF FRIGIDITY are concerned only with *nullity of marriage* and *divorce*.

STERILITY

Sterility in the sexual/reproductive context is defined as *the incapability of fertilisation or reproduction*. However, the term "sterility" has fallen out of favour, and has been replaced by the phrase "absolute infertility." Thus, **absolute infertility** in the **male** is the *inability to fertilise the ovum*. Impotence and absolute infertility may co-exist, but not invariably - a person may be potent but sterile; conversely, he may be impotent, but not necessarily sterile. Absolute infertility in the **female** is her *inability to conceive, due to inadequacy in genital structure or function*.

Absolute infertility is complete and usually irremediable. On the other hand, relative infertility, which is the diminished capacity to produce offspring, can often be corrected. A medical jurist's opinion is usually sought only in cases of absolute infertility (either male or female). Opinions regarding relative infertility are better given by practising genitourinary surgeons or obstetricians/gynaecologists.

CAUSES OF ABSOLUTE INFERTILITY IN THE MALE

The factors essential for fertilisation of the ovum are normal spermatogenesis, transport of the spermatozoa to the external urethral meatus, and deposition of these spermatozoa in the female genital tract (insemination). Taking these factors into consideraton, the causes of absolute infertility in the male (excluding the physiological infertility that exists before puberty and in extreme old age) can be classified as[4]:

1. **AZOOSPERMATOGENIC** (Total failure to produce living spermatozoa) :
   a. *Seminiferous tubular necrosis*, e.g., Klinefelter's syndrome
   b. *Germinal aplasia*, either idiopathic, or due to the XYY syndrome, or exposure to radiation/drugs;
   c. *Arrest of maturation*, as in varicocoele, and
   d. *Endocrinopathies*.

2. **OLIGOSPERMATOGENIC** (Failure to produce living spermatozoa in sufficient numbers):
   a. *Congenital conditions*, e.g., Frohlich's and Klinefelter's syndromes, primary testicular failure, and cryptorchidism, or
   b. *Acquired conditions*, e.g., general debilitating diseases, local diseases such as adolescent mumps orchitis/ tuberculous epididymo-orchitis/varicocoele/carcinomata, as an occupational hazard in stokers/athletes/technicians exposed to radiation, in endocrinopathies, and drug-induced (nicotine, alcohol, cannabis, cocaine).

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3. **ASTHENOSPERMATOGENIC** (Defects in sperm movement):
   a. *idiopathic*
   b. *spermatozoal structural defects*
   c. *antisperm antibodies*
   d. *genital tract infections*
   e. *prolonged abstinence*

4. **NORMOSPERMATOGENIC**: Infertility may be because of:
   a. *structural/functional abnormalities in the male*
   b. *abnormal coital habits* (including poor timing)
   c. *acrosomal defects in the spermatozoa*
   d. *immunological factors*
   e. *unexplained causes*

5. **ABSENT EJACULATION** due to surgery (e.g., vasectomy), vascular occlusion, diabetes mellitus and psychological disturbances, or may be drug-induced.

CAUSES OF ABSOLUTE INFERTILITY IN THE FEMALE[5-7]

These may be due to organic defects (either congenital or acquired), hormonal dysfunction, or chromosomal abnormalities.

1. **ORGANIC CAUSES**:
   a. *Congenital defects*:
      i. of the **vagina**, which may not have developed at all, may not be canalised, may be septate, or may manifest an imperforate hymen.
      ii. of the **cervix**, which may be elongated or conical, or may have a pinhole os (hostile cervical mucosa may also obstruct/impede penetration of the ovum by the sperm).
      iii. of the **uterus**, which may be either absent or retroflexed.
      iv. of the **fallopian tubes**, which may be stenosed (an extremely rare condition).
   b. *Acquired causes*:
      i. diseases of genital tract, e.g., gonorrhoea leading to salpingitis.
      ii. diseases of ovaries, uterus or fallopian tubes.
      iii. exposure to radiation.
      iv. exposure to drugs.

2. **HORMONAL DYSFUNCTION** leading to menstrual irregularities may occur from pituitary dysfunction, adrenogenital syndrome, or prolonged use of contraceptives.

3. **CHROMOSOMAL ABNORMALITIES**, e.g., Turner's syndrome.

Apart from the causes of absolute infertility in the male and female enumerated above, there is a host of unexplained and unexplored factors in both males and females, which are beyond the scope of this handbook.

MEDICOLEGAL ASPECTS OF STERILITY (ABSOLUTE INFERTILITY)

Civil Cases

- The putative father, in a suit of disputed paternity, may plead that he is sterile, and so could not have fathered the child.
- A patient may claim damages for loss/diminution of reproductive capability as a result of professional negligence during a surgical operation.
- Absolute infertility in the male is the prime indication for artificial insemination (donor) or adoption of a child.

Criminal Cases

Battery or criminal negligence may be cited as the cause for the incapability of a male for fertilisation, or of a female for conception.

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INVESTIGATION OF A CASE OF STERILITY (ABSOLUTE INFERTILITY)

1. In both **males** and **females**, a detailed physical and marital history, general examination, and scrutiny of secondary sexual characteristics are mandatory. A psychological assessment may also be carried out when indicated.

2. In the **male**, the specific investigations to be conducted include:
   a. *initial basic laboratory evaluation*
      i. of seminal fluid for its physical characteristics, morphology and motility of spermatozoa, and presence and level of fructose; and
      ii. of hormonal levels with particular reference to follicle-stimulating hormone (FSH), gonadotropin and gonadotropin-releasing hormone (GnRH), testosterone and prolactin, and luteinising hormone (LH);
   b. *transrectal ultrasonography* (TRUS) to detect absence or anatomical abnormalities of the seminal vesicles or ejaculatory ducts; and
   c. *second-level testing* (not all patients need these):
      i. to rule out antisperm antibodies,
      ii. leucocyte staining of semen,
      iii. testicular biopsy,
      iv. vasography, and
      v. functional evaluation of spermatozoa.

3. In the **female**, the specific investigations to be conducted include[8]:
   a. *evaluation of ovulation* is extremely important. Its adequacy is examined by several methods, including basal body temperature charting, and testing of urinary LH, midluteal progesterone levels, endometrial biopsy, and ultrasonic monitoring of the development and disappearance of the ovarian follicles. However, combinations of these methods must be used over several cycles before an opinion can be passed with any degree of accuracy.

   b. *evaluation of the fallopian tubes* is equally important. The usual investigation is the hysterosalpingogram using either oil- or water-based dyes, to determine the patency of the fallopian tubes as well as the shape of the uterus. It is generally performed in the early follicular phase of the cycle, once menstrual bleeding ceases.

   c. *laparoscopy* may be indicated when clinical examination or prior investigations have raised a suspicion of pelvic pathology, or when all other investigations have proved normal.

   d. *additional investigation* of the functional relation between spermatozoa and ova may be performed when considered absolutely necessary.

ARTIFICIAL INSEMINATION

Having considered impotence and absolute infertility, it would be appropriate to know how a woman may be enabled to conceive by artificial means. The oldest and simplest of these is **artificial insemination**, defined as *the introduction of semen into the vagina other than by coitus.* The first reported usage of this method was by **John Hunter**, at the end of the **13th century**, on the wife of a linen draper; a normal pregnancy ensued.

The procedure employs a sterile syringe to introduce 1 millilitre of semen near or into the cervix at or about the time of ovulation, since that is the only time when the method would prove effective. The semen should be collected by masturbation into a widemouthed sterile glass container, after 3 days' abstinence. The specimen should be used within 2 hours of collection, during which it should be sub-

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jected to examination to ensure its normalcy with respect to its physical characteristics and the quality of spermatozoa (number, morphology, and motility).

If the semen used for this purpose is derived from the woman's husband himself, the procedure is called *homologous artificial insemination* (AIH), whereas if it is obtained from a person other than the husband, the method is termed *heterologous artificial insemination*, or, for differentiation from the first type, *artificial insemination (donor)* (AID). The use of frozen semen stored in sperm banks for heterologous artificial insemination is becoming increasingly popular, as is pooling of the husband's semen with that of a donor (which is psychologically advantageous to both the husband and his wife).

The **indications** for artificial insemination are

1. for AIH: an impotent but fertile husband; and
2. for AID:
   a. a sterile husband,
   b. genetic defects in the husband, carrying a high risk of mortality, which would definitely be transmitted to the child (e.g., blood dyscrasias), and
   c. Rh incompatibility between husband and wife, which would result in erythroblastosis foetalis in the child.

Homologous artificial insemination does not engender any significant legal or ethical problems, so no special precautions are called for. On the other hand, when AID is contemplated, the registered medical practitioner who undertakes the procedure must observe with greatest care, the following precautions in the selection of the donor, lest he be accused of ethical malpraxis :

1. The expressed written informed consent of both the recipient and her husband, and of the donor and his wife, is mandatory.
2. Absolute confidentiality must be maintained, i.e.
   a. neither should the identity of the donor be revealed to the recipient, her husband, or their offspring,
   b. nor should the identities of the recipient, her husband or the child be made known to the donor (or, should he be married, to his wife or children).
3. The donor
   a. should be less than 40 years of age,
   b. should not be related by blood to either the recipient or her husband (however, it is preferable that he resemble the husband physically, socially, and intellectually as closely as is possible),
   c. should be in sound physical and mental health, and should be free of hereditary, familial, and sexually transmitted diseases, and
   d. should be potent and fertile (if married, he should have at least two healthy children of his own).

MEDICOLEGAL ASPECTS OF ARTIFICIAL INSEMINATION

The medicolegal aspects of artificial insemination vary from country to country. Although practised widely in the West, it has not gained much popularity in India (except in veterinary practice). Strangely, even abroad, it has no definitive legal sanction in statutory or case law. Thus, multiple legal questions arise about the procedure:

1. Does artificial insemination (donor)
   a. amount to rape or adultery?
      Not in India or in the United Kingdom, where sexual intercourse is necessary to constitute the offence.
   b. constitute a valid ground for
      i. Divorce by reason of adultery? Again, this is not a valid reason (because of the definition of the offence), unless the procedure was performed without the expressed written informed consent of the husband.

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ii. Annulment of marriage by reason of non-consummation?

This question would definitely arise if the husband was both impotent and sterile prior to the marriage. However, no such instance seems to have been reported to date, for obvious reasons.

2. Is a child born of artificial insemination legitimate? Peculiarly, the law says "No", regardless of whether the child was born of AIH or AID! This view raises further queries, such as
   a. If the husband is not the father of the child, then who is? Or is the child to be considered fatherless? This is an anomaly, because no child can be begotten without a father (at least, until the advent of cloning!).
   b. Would naming the husband of the child born of AID as its father in legal documents (birth certificate, applications for various purposes, etc.) amount to a false declaration?
   c. How could the child lawfully inherit the property of the "father"?

   The general consensus of opinion as to the above question of **legitimacy** and its corollaries is that the child born of artificial insemination must be legally adopted by its parents, so that
   i. the husband is legally considered as the father of the child, even though he may not have contributed to its conception, and
   ii. the child can lawfully inherit the property of its parents, or be entitled to maintenance should its parents divorce.

3. Does sexual intercourse between a child born of AID and the children of the donor born in wedlock amount to incest?
   Theoretically it would, but practically, the probability of such an event is infinitesimal.

Finally, it must always be kept in mind that the physician who conducts an artificial insemination can be held liable for civil negligence, indecent assault, or breach of trust, unless
1. he exercises the utmost skill and care in the selection of a donor, and in the performance of the procedure,
2. he ensures that a registered nurse is present throughout the procedure,
3. he himself conducts the delivery of the child conceived by the procedure which he has performed, and
4. he maintains all records pertaining to the procedure meticulously and in the strictest confidence.

OTHER METHODS OF ASSISTED REPRODUCTION

This chapter would be incomplete if the various recently-evolved techniques of assisted reproduction (ARTs) were not at least enumerated. In chronological order, these are

1. ***in-vitro fertilisation and embryo transfer*** (IVF-ET), first successfully employed by **Steptoe** and **Edwards** in **1978**,[9] which consists of controlled ovarian hyperstimulation (COH) using one or more ovulation-inducing drugs, aspiration of oocytes, insemination of these oocytes in vitro, and transfer of the cleaved embryos back into a woman's uterus. Successful pregnancy and delivery rates vary from clinic to clinic, and are also dependent upon the woman's age, but approximate 18 per cent and 14 per cent, respectively.

2. ***gamete intra-fallopian transfer*** (GIFT), an option used only in patients who have at least one patent fallopian tube, and whose infertility is longstanding and not responding to other measures. This method involves COH, aspiration of oocytes by laparotomy or

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minilaparotomy, and transfer of oocytes and sperm back into the fallopian tube. The technique was developed by **Asch** et al who reported it in 1985.[10] Pregnancy rates with this technique vary between 30 and 35 per cent (these decline with advancing age), and delivery rates between 23 and 25 per cent.

3. ***pronuclear stage tubal transfer*** (PROST), another option only for those with at least one normal fallopian tube. In this procedure, originally used by **Devroey** et al in 1989,[11] COH is followed by ***ultrasound-guided egg aspiration*** (USGEA), and fertilisation in vitro. The eggs are observed 24 hours after insemination for the development of pronuclei as proof of fertilisation. If these are identified, three or more zygotes are laparoscopically transferred into the fallopian tubes. Pregnancy rates as high as 45 per cent have been reported, but the delivery rate does not seem to have exceeded 17 per cent per procedure.

4. ***micromanipulation techniques*** to assist fertilisation,[12] which have been rapidly developed during the past several years as adjuncts to the ARTs already described. These are attempted predominantly in those infertilities associated with the male factor as a cause or component. Those presently in use are ***zona drilling*** (**Gordon** and **Talansky, 1986**), ***partial zona dissection*** (PZD, **Cohen, 1989**), ***perivitelline sperm transfer*** (PST, **Lasalle** et al, 1987), and ***intra- cytoplasmic sperm injection*** (ICSI, **Bongso** et al, **1989**).

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CHAPTER - 17

Virginity, Pregnancy and Delivery

"Virginity is like wine, becomes better with age upto a point, beyond that it becomes mildewed".

- Samantha Davis, (Hollywood starlet)

VIRGINITY[1]

Virginity, also referred to as **chastity**, is defined as "the state of being *virgo intacta*, i.e., one who has never had sexual intercourse." In contrast, **defloration** refers to the "deprivation of virginity; the rupture of the hymen, either in coitus or in vaginal examination." (These terms were formerly applied only to females, and only with respect to penile-vaginal sexual intercourse, but now describe penile-anal sexual intercourse in both females and males as well.)

MEDICOLEGAL ASPECTS OF VIRGINITY

1. In **Civil cases**, whether a woman is a virgin or not may have to be decided in instances of:
   a. **nullity of marriage**, where virginity is proof of *non-consummation*,
   b. **divorce**, when deliberate withholding of a non-virgin state before marriage may be cited as grounds, and
   c. **defamation of character**, when a woman brings an action for tort for illegal damage to reputation, against a person who has alleged that she is not a virgin.

2. In **Criminal cases**, loss of virginity may be offered as an *index* of sexual intercourse to substantiate a charge of rape.

SIGNS OF VIRGINITY

These may be considered under two heads : **extra-genital** (i.e., in the **breasts**) and **genital**. It is important to be aware here of the condition of ***false virginity***, in which the hymen (*vide infra*) may persist even after repeated coitus. The signs of true virginity, by which it can be differentiated from false virginity, are presented in **Table 17.1**.

THE HYMEN AND ITS MEDICOLEGAL IMPORTANCE

The hymen is a thin (but firm) fold of connective tissue, covered by mucous membrane, about 1mm in overall thickness, situated at the vaginal orifice. Commonly, it is deficient anteriorly, and usually has a central aperture, but may be **annular, semilunar, cribriform, septate, subseptate, fimbriate, infundibuliform** (funnel-shaped), **imperforate,** or may even be **congenitally absent**.[3] Embryologically, the hymen is the remnant of the vaginal plate that develops between the Mullerian ducts and the cloaca; it has no useful function.[4]

CAUSES OF HYMENAL RUPTURE

1. As a result of sexual intercourse, the hymen is

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usually torn posteriorly, with unilateral or bilateral tears extending to the point of its attachment to the vaginal wall. These ruptures bleed freely when fresh, and heal by scar formation. After the delivery of a child per vaginum, the hymen is almost completely torn, leaving only small remnants called "carunculae myrtiformes" or "carunculae hymenalis".

2. In a true virgin, the hymen may be ruptured because of [5,6] :
   a. **trauma**, due to
      i. an accidental fall on a protruding substance/object (This is not usually possible by strenuous physical exercise or by merely forcible separation of the thighs.)
      ii. instrumental masturbation with large foreign bodies (provided the instrument or object enters deep enough. Digital masturbation, unless practiced with two or more fingers, does not usually rupture the hymen.)
      iii. careless insertion of sanitary tampons
      iv. surgical operations for imperforate hymens, and per vaginal examinations on virgins (These latter are absolutely contraindicated unless there is no alternative.)
      v. the introduction of foreign bodies for reasons other than those described above, e.g., the insertion of sola pith into the vagina of very young girls to render them suitable for sexual intercourse

   b. **ulceration** as a result of disease (especially diphtheria). In such cases, the entire hymen is destroyed leaving only scar tissue.

Besides being of limited anatomical value, the hymen, from a medicolegal viewpoint, is a grossly overrated piece of tissue - even experts can never be certain of virginity because of false virginity and hymenoplasty (surgical repair). In this latter condition, however, the hymen is usually taut, has an unusually narrow aperture, and exhibits fine scars at the sites of repair of previous tears.

PREGNANCY (gestation, foetation, gravidity)

Pregnancy is the physiological condition of having a developing embryo or foetus in the body, from the time of fertilisation of an ovum by a spermatozoon until the birth of a child. Since it is not possible to determine the precise moment at which the spermatozoon fertilises the ovum, so also the exact moment of commencement of pregnancy is uncertain. About 7 days after fertilisation, the ovum reaches the uterine cavity and implants itself in the uterine wall.

The mean duration of a normal pregnancy is usually 266 days (38 lunar weeks) from the day of ovulation, or 280 days (40 lunar weeks) from the first day of the last menstrual period.[7] In obstetric practice, it is customary to estimate the expected date of delivery by adding 9 calendar months and 7 days to the first day of the last menstrual period (**Naegele's rule**)[8]. If the duration of pregnancy is calculated by this rule, it is obvious that ovulation and fertilisation follow the "onset" of pregnancy by about 2 weeks. Therefore, while both obstetricians and the law describe the duration of pregnancy in terms of gestational or menstrual age, embryologists and other histologists prefer to go by ovulatory or fertilisational age. It is also customary to divide the duration of pregnancy into "trimesters", both to facilitate ease of diagnosis and awareness of the problems particular to each. (The latter are beyond the scope of this handbook.)

MEDICOLEGAL ASPECTS OF PREGNANCY

Civil Cases

- Malicious persons may allege either verbally (*slander*) or in writing (*libel*), that an unmarried woman or widow is pregnant, in which case

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Table 17.1

FEATURE	TRUE VIRGINITY	FALSE VIRGINITY
a. Extragenital signs (in the breasts):		
i) Shape & consistency	Hemispherical and firm	Pendulous*
ii) Areolae	Pink	Pigmented, with Montgomery's tubercles
iii) Nipples	Small, and pink in colour	Enlarged and pigmented*
		**These are significant only if pregnancy has resulted. Even then, more weightage is given to the nipples and areolae than to shape & consistency*
b. Genital signs		
i) Labia majora	Firm, and lie in apposition	Separated and flabby
ii) Labia minora	Pink; soft and sensitive to the touch	Elongated, brownish; separate and flabby; not sensitive to the touch
iii) Clitoris	Small	Enlarged
iv) Fourchette & posterior commissure	Intact	Torn
v) Vestibule	Narrow	Wide
vi) Vagina	Narrow (H-shaped on cross-section); walls firm; rugosity easily discernible	Enlarged and roomy; walls yielding; rugosity ironed out
vii) Hymen	Intact; membranous; admits only the tip of the little finger through the orifice painfully	Loose/folded/elastic, thick, tough and fleshy, or relaxed, undulant and permits depression; easily admits two fingers through the orifice

she would definitely want to vindicate herself.

- A concealed pregnancy before marriage, or after marriage by a person other than the lawfully wedded spouse, would constitute a valid ground for divorce.
- The duration of pregnancy assumes importance when the legitimacy of a **posthumous child** (one born after the death of its father) is questioned.

Criminal Cases

- Pregnancy is positive proof of sexual intercourse in a trial for rape.
- Pregnancy may be a motive for the suicide of a married or unmarried woman, or for the murder of a married woman by her husband, when it is the result of an illicit sexual relationship. Such a situation has also driven women to criminal abortion and to foeticide or infanticide.

Civil and Criminal Cases

A woman may feign pregnancy (a condition termed **pseudocyesis**, or false/imaginary pregnancy) in order to

a. avoid capital punishment (Because the unborn

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child is innocent of any crime its mother may have committed, and must not be punished for it, the death sentence is either commuted to imprisonment for life, or is postponed till after the delivery of the child)

b. to compel a person to marriage.

c. to secure more maintenance in a case of divorce.

Pseudocyesis is also encountered in sterile or infertile women who long for children.

Also to be considered here are the conditions of **superfoetation** (where two or more pregnancies result from the fertilisation of two or more ova of *different* ovulatory periods), and **superfecundation** (in which two or more ova of the *same* ovulatory period are fertilised by *separate* acts of sexual intercourse with the same person, or two different persons). Although superfoetation is theoretically possible, it has not been demonstrated beyond doubt in human females (though it is known to occur in certain lower animals, e.g., mares). On the other hand, superfecundation is not only a more likely possibility, but has also been described in literature. The earliest written report of this phenomenon was by **John Harris** (the first physician in America to earn a medical degree) in **1810.** Other descriptions, much later, are by **Harris (1982)**, and **Terasaki et al**, who used HLA typing to establish that dizygotic twins were the offspring of different fathers.[13]

DIAGNOSIS OF PREGNANCY[7]

In all the cases listed above, the woman in question may be sent to a registered medical practitioner for examination and certification as to whether she is pregnant or not, (the expressed written consent of the woman, duly witnessed, is mandatory.) To perform this duty, one must be familiar with the symptoms and signs of pregnancy, as well as the laboratory and other examinations that are useful in the diagnosis of pregnancy. These may be classified under three heads : **presumptive, probable** and **positive.**

Presumptive Evidence of Pregnancy

is based largely on the signs exhibited and symptoms experienced by the woman.

a. The **signs** are:

i) **secondary amenorrhoea**, which, though strongly suggestive of pregnancy, is not reliable until 10 or more days have passed after the expected date of the first day of menstruation. It must be remembered that:

- there are rare instances of pregnancy in women who have never menstruated or who are supposedly menopausal,
- that menstruation may continue even after conception, and
- that there are several causes for amenorrhoea other then pregnancy, e.g., environmental, nutritional, organic diseases such as severe anaemia and tuberculosis, and psychic causes.

ii) **mammary changes**, more marked in primigravidae (women who are pregnant for the first time):

- progressive enlargement and tenderness.
- easily visible superficial veins.
- in the second month, hyperpigmentation of the areolae and nipples, with the appearance of *Montgomery's tubercles* (raised spots due to enlargement of the sebaceous glands).
- from the fourth month onwards, *colostrum*, which consists of free fat globules and large phagocytic cells filled with droplets of fat, can be squeezed from the breasts. (All these changes can also be seen in tumours of the ovary which secrete prolactin, women who take tranquilisers which induce prolactinaemia, women who suffer from psychic pseudocyesis, and after repeated stimulation of the breasts.)

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iii) **changes in the vagina** :
- increased secretion of mucus.
- palpable pulsation of the vaginal arteries.
- **Jacquemir's/Chadwick's sign,** i.e., bluish discoloration of the vaginal mucosa (This is not a reliable sign, since it can be caused by anything that obstructs proper venous return), and
- **Barnes' sign,** i.e., flattening of the anterior vaginal wall because of upward tilting of the cervix.

b. The **symptoms** experienced by a woman during early pregnancy include:
i) **Morning sickness,** which refers to the nausea, often followed by vomiting, soon after waking, and which usually disappears on its own later on during the pregnancy. (Like many other presumptive symptoms, this is not to be relied on because it is seen mostly in primigravidae, may not occur in all cases, and may also be a symptom of several gastrointestinal disorders). It may occasionally persist as *hyperemesis gravidarum*.
ii) **Disturbances in micturition,** such as increased frequency during the first trimester, a return to normal as the uterus grows into the abdomen, and reappearance of the increased frequency at or near the end of term because of the descent of the foetal head into the pelvis.
iii) **Easy fatiguability, hypersalivation, unusual appetite, and increased irritability.**

Probable Evidence of Pregnancy
can be obtained from various signs, symptoms, and the results of laboratory investigations.

a. The **signs** are:
i) **abdominal changes** :
- progressive enlargement after the 12th week, so that the umbilicus becomes flush with the skin,
- increased pigmentation (also seen over the pubis and axillae), with *linea albicantes*, (these lines, and progressive enlargement are merely signs of abdominal distension.),
- *striae gravidarum* (more pronounced in primigravidae), and *linea nigra* (which are again not diagnostic because they may not be seen, or may also be seen in women who regularly take combined contraceptives).

ii) in the **cervix**:
- mucus becomes a thick and viscid plug, blocking the cervical canal.
- **Goodell's sign** (softening because of increased vascularity) can first be appreciated at 6 to 8 weeks. (The cervix becomes more and more patulous as pregnancy advances, but this change may also occur in acute cervicitis, haematometra, and in women who are longterm regular users of combined contraceptives. Towards term, there is apparent shortening of the cervix, the cervical canal becomes circular, and the external os admits the tip of a finger with ease and to a greater depth).

iii) **uterine souffle** (a soft, blowing murmur, synchronous with the maternal pulse, which can be heard with a stethoscope applied just above the midline of the inguinal ligament) is usually audible from the 16th week onwards.

iv) in the **uterus,** apart from progressive enlargement, the following can be elicited:
- **Hegar's sign** (softening and easy compressibility of the lower uterine segment) first appears at 6 to 8 weeks. (This sign is elicited by introducing two fingers of one hand into the posterior fornix while the other hand rests on the pelvis just above the pubic symphysis. On pressing upwards with the first hand, it seems that

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there is no intervening uterus between the two hands).

- **Braxton-Hicks' sign,** i.e., the appreciation of intermittent painless contractions of the uterus from the 16th week onwards by palpation of the abdomen, occurring at intervals of 5 to 20 minutes, each lasting for about 1 to 5 minutes, and

- **Ballottement** (tossing of a ball), i.e., the method of eliciting the presence of the foetus floating in the liquor amnii. This could be determined either **externally** or **internally.** *External ballottement* is elicited by placing the palms of both hands over the abdomen on either side of the midline, and pushing firmly with one hand towards the other, whereupon the impetus thus given to the foetal head can be felt against the other. *Internal ballottement* is done by inserting two fingers of one hand into the posterior fornix and pushing sharply upwards. The foetal head will first move upwards in the liquor, then settle down onto the fingers. (It must be kept in mind that ballottement cannot be elicited if the liquor amnii is deficient, and that soft subserous myomata can be mistaken for the foetal head.)

b. The only **symptom** that could be taken as probable evidence of pregnancy is **quickening,** i.e., the first perception of foetal movements by the mother. This is first possible in the 16th to 20th week, and increases in intensity as pregnancy advances.

c. **Laboratory investigations** for pregnancy all depend upon increased levels of **human chorionic gonadotropin (HCG)** in the urine, using either **biological** or **immunological methods.** The morning midstream urine of the woman suspected to be pregnant is used for these tests.

i) The **biological (animal) tests,** which are easy to perform but time-consuming, include
   - the **Aschcim-Zondek** (female mouse) **test**
   - the **Friedman** (female rabbit) **test**
   - the **Hogben** (female toad or frog) **test** and
   - the **Galli-Mainini** (male toad) **test**

   All these tests are, however, obsolete and only of historical interest, having given way to

ii) The **immunological tests,** which are much faster, more specific, less cumbersome, more sensitive, and do not require laboratory animals, equipment or special skills.
   Taking advantage of the antigenic properties of HCG, the examples include
   - the **haemagglutination** (Prognosticon) **test**
   - the **latex-aglutination** (Gravindex) **test,** which could deliver a result in two hours, and its improvement, the **Pregcolor test,** which can be read in **two minutes**
   - **radioreceptor assay,** which is highly sensitive and 98 per cent accurate.

   However, all these HCG-based tests will also be positive in those conditions in which abnormal or pathological products of conception are formed, viz., hydatidiform mole and chorionepithelioma. Further, false positives could also occur because of the biological and immunological similarities between HCG and luteinising hormone (LH).

Positive Evidence of Pregnancy

Absolute/certain/conclusive/sure signs can be grossly appreciated only from the 4th to 5th month (16th to 20th week) of gestation onwards. They include:

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a. **Foetal parts and foetal movements,** discernible by bimanual palpation (This may be difficult when the abdominal wall is fatty, or in hydramnios).

b. **Foetal heart sounds,** with a normal rate of 120 to 160 per minute, which sound like the muffled ticking of a watch , variable in site. These can be heard on auscultation of the abdominal wall with a foetoscope. (They must be distinguished from those of the mother, sounds caused by the movements of the foetus, and sounds produced by the passage of liquids and gases through the mother's intestines). Foetal heart sounds can be detected much earlier if more advanced techniques are used, e.g., at 10 to 12 weeks by Doppler principle with ultrasound, at about 8 weeks by pulse-echo and real-time sonography, and even as early as 7 weeks by echocardiography.

c. **Radiological demonstration of the foetal skeleton,** which, although possible in some cases at the 20th week of pregnancy, is contraindicated during the first trimester. (By ultrasonography, a small white gestational ring can be seen as early as the 6th week of gestation, and distinct echoes from the embryo within this gestational ring can be discerned by careful scanning 2 weeks later).

DELIVERY

Delivery is defined as *complete evacuation of a foetus (that has attained viability) and other products of conception from within the uterus and genital passages into the external environment.*[1] This process may be spontaneous or assisted. When delivery occurs after the full period of gestation, i.e., 280 days, it is called a "full -term delivery". If it occurs earlier or later than this, it is said to be "preterm/premature" and "post-term/postmature" respectively.

MEDICOLEGAL ASPECTS OF DELIVERY

1. In **civil cases,** whether a woman has delivered or not needs to be determined in instances of:
   a. **Feigned delivery,** when a woman may pretend to have been pregnant for some time and later produce a child, alleging that it is hers to claim property of a deceased husband. Such a child is called a **Suppositious child.**
   b. **Affiliation cases** (affiliation = adoption), in which a woman claims that her child has been fathered by a person who is not her lawfully wedded spouse, or by a husband who has subsequently divorced her, and who must therefore adopt the child as his own and pay for its upkeep.
   c. **Contested legitimacy of a child,** when, as part of the evidence that a child was born of a particular woman and her husband, it must be proven that the woman did indeed deliver a child at the time claimed by her.

2. In **criminal cases,** the question of delivery assumes importance in trials for
   a. **abortion** and **infanticide,**
   b. **concealment of a child** begotten out of an illicit sexual relationship, and
   c. when an **affiliation case** proceeds to the degree of **blackmail.**

Forensic medicine is therefore concerned with the determination of recent and remote delivery in both the living and the dead. This is best achieved during the **puerperium,** which is literally defined as the period of confinement of a pregnant woman during and just after the birth of a child.

SYMPTOMS AND SIGNS OF RECENT DELIVERY IN THE LIVING (at full term)[7]

1. The **symptoms** include:
   a. **Fatigue,** which is most pronounced immediately after delivery, and more so when labour has been prolonged.

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b. **Loss of weight** because of both expulsion of the contents of the uterus and
c. **Diuresis** during the 2nd to 5th days, because of physiological reversal of the increased intracellular fluid during pregnancy.
d. **Afterpains** (experienced by primiparae as tonic contractions, and by multiparae as vigorous contractions, at intervals. They are occasionally painful and/or severe, beccome mild after 3 days, and usually disappear after 7 days, but may last longer in some patients).
e. A sharp **rise in temperature** for the first 24 hours postpartum (*milk fever*) - any fever which lasts longer is most likely due to infection,
f. **Transient depression**, a common feature which is usually self-limiting and responsive to treatment or reassurance, but may progress to **puerperal psychosis**, as a result of which the mother may neglect, ill-treat, or even kill her child.

2. The **signs** of recent delivery are seen
   a. In the **breasts**, similar to those of pregnancy, but more marked (**Colostrum** can be squeezed out for the first 2 days, after which it is replaced by mature milk).
   b. In the **abdomen**, which remains soft and flabby for several weeks (although tone and contour can often be regained by exercise), displays silvery-white **linea albicantes**, and, when Caesarean section has been performed, the operative incision in the lower segment.
   c. In the **vagina and its outlet**, which may display the following features :
      i) **Smooth-walled, relaxed and wide** (The width progressively decreases, but nulliparous dimensions are never regained).
      ii) **Ironed-out rugae** (These reappear by the 3rd week in primiparae, but never in multiparae).
      iii) **Lacerated walls** which heal rapidly.
      iv) **Carunculae myrtiformes** of the hymen.
      v) The **labia** in primiparae, appear swollen, tender, gaping, bruised and/or lacerated.
      vi) The **perineum**, which shows ruptures of the fourchette and posterior commissure with or without a sutured/healing incision of episiotomy, and
      vii) **Lochia**, i.e., an alkaline discharge with a peculiar, sour, disagreeable odour, consisting of red blood cells, leukocytes, shreds of decidua, epithelial cells and bacteria. (It is occasionally foul-smelling, but this is merely suggestive of infection, not proof). The appearance of this discharge changes as the puerperium progresses, so that
         - for the first few days, it is red (*lochia rubra*),
         - from the 5th to 10th days, it is watery and pale (*lochia serosa*),
         - from the 10th day onwards, it becomes thicker, scantier, and white or yellowish-white (*lochia alba*), and
         - it disappears in 2 to 3 weeks.
   d. In the **cervix** (which is thin, collapsed and flabby soon after delivery) :
      i) the **external os**
         - shows lacerations of its outer margins (These heal rapidly to leave permanent bilateral depressions), and
         - contracts slowly, so that for the first 2 days, it admits 2 fingers easily, and at the end of 1 week, 1 finger with difficulty, and
      ii) The **walls** simultaneously thicken to re-establish the cervical canal, and
   e. In the **uterus** : because of *involution*,
      i) From the 1st to 3rd days after delivery, the fundus of the uterus is midway between the umbilicus and the symphysis pubis,

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ii) Within 2 weeks, it descends into the true pelvis (and cannot be felt above the symphysis pubis),

iii) In 5 to 6 weeks postpartum, it attains its normal nonpregnant size (**Table 17.2**).

2. **Laboratory investigations**: The **biological tests** for pregnancy are positive for about 7 to 10 days after delivery.

SIGNS OF RECENT DELIVERY IN THE DEAD

Besides observing the gross changes in the breasts, abdomen, the vagina and its outlet, and the cervix, and palpating the uterus per abdomen, the **uterus, ovaries** and **peritoneum** are also examined.

1. a. The weights and dimensions of the involuting uterus are listed in **Table 17.2**.
   b. Morphology of the involuting uterus :
      i) If lower segment caesarean section has been performed, its fresh or healing incision will be seen.
      ii) *Cut section* soon after delivery shows
         - anterior and posterior walls each 4 to 5 cm thick
         - a body made up mostly of myometrium, lined by basal decidua
         - a placental site about the size of a palm, which rapidly decreases in size and
         - an ischaemic appearance because the vessels are compressed by the contracting myometrium.

      iii) *Cut section* 2 to 3 days after delivery shows that the remaining decidua has differentiated into two layers : a superficial layer which is necrotic (shed in lochia) and a basal layer, which is the foundation of a new endometrium.

      iv) *Cut section* 2 weeks after delivery shows
         - that the rapidly growing endometrium has covered the entire surface except for the placental site, and
         - a placental site 3 to 4 cm in size.

      v. At 6 weeks after delivery, even the placental site gets covered by new endometrium.

   c. The ovaries and Fallopian tubes are congested. On microscopic examination, an intact or degenerating corpus luteum can be seen.

   d. i) The peritoneum covering most of the uterus is folded and wrinkled, and
      ii) the broad and round ligaments are lax ; they take a long time to recover their pre-pregnancy consistency.

Table 17.2 Weight and dimensions of involuting uterus

Duration after delivery	Weight	Dimensions
Freshly delivered	1000 to 2000 g.	Variable
Immediately after delivery	Variable, but less than above	22 cm x 15 cm
2 to 3 days postpartum	Variable, but still less	Less than above
End of the 1st week	500 g.	13 to 15 cm x 8 cm
End of the 2nd week	300 g.	10 cm x 5 cm
Soon thereafter	100 g. or less	Variable, but less than above
5 to 6 weeks	Normal nonpregnant weight	Normal nonpregnant size

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SIGNS OF REMOTE DELIVERY IN BOTH THE LIVING AND THE DEAD

These include the permanent morphological alterations that have been previously described, with linea albicantes of the breasts and slight concavity inwards of the inner surface of the uterus. If there is any doubt from naked-eye appearances as to whether delivery has taken place or not, the endometrium must be examined under the microscope for the presence of trophoblastic endothelium and chorionic villi.

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CHAPTER - 18

Abortion

"The trauma of a pregnancy resulting from rape is not merely physicel. We must consider sympathetically the damage it can wreak on a sensitive mind".

- Dr. Alec Bourne (1938)

DEFINITION

Abortion is defined as *the premature expulsion of products of conception from the womb, at any time before its period of gestation is complete*. The word 'abortion' is derived from the Latin word 'aboriri', which means "to get detached from the proper site".[1] By convention however, **abortion** is the term used to denote termination of pregnancy during the first trimester, while that occurring in the second trimester is called **miscarriage,** and that occurring in the last trimester of pregnancy is called **premature birth.**

CLASSIFICATION

Abortion is classified into two types : **natural** and **artificial.** Natural (spontaneous) abortion occurs in the first trimester of pregnancy. Artificial abortion is that which is deliberately induced. The latter is further subdivided into *justifiable* (therapeutic) and *criminal.*

Therapeutic and Criminal Abortion: Before medical termination of pregnancy became a legal option, women were not allowed to abort, unless there was a proven threat to life. Those women who were denied legal abortion (unmarried females, widows and rape victims), therefore had to resort to alternative means of abortion i.e. criminal abortion.

Nowadays, criminal abortion has come to mean the termination of pregnancy by the woman herself, or by another party not qualified to perform the termination, or when it does not conform to the MTP Act of 1971. The liberalisation of rules relating to the performance of abortions in the western world owes much to **Dr. Alec Bourne.** In the year 1938, a fourteen-year-old girl was enticed into the stables near a beach by some lifeguards, and then raped. As a result of this she became pregnant. Dr. Alec Bourne, a practicing gynaecologist of impeccable credentials in suburban London, performed an abortion with the consent of the parents. He did so without any secrecy, in a reputed London hospital. Later on, he notified the Police, making a full and clear disclosure. A case was filed against Dr. Bourne. In the trial he justified his action on the grounds that the girl would have been carrying around in her body, for nine months, a child conceived out of rape, which would have harmed her mental health. It was for the first time that mental trauma arising out of an unwanted pregnancy was highlighted in a court of law. Dr. Bourne was acquitted. This has since become a benchmark case, serving as a forerunner for the subsequent liberalisation of medical termination of pregnancy.

THE MEDICAL TERMINATION OF PREGNANCY ACT (1971 Amended 1975)

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The **MTP Act** of 1971 has made provisions for allowing abortions in India on the basis of clearly specified guidelines. This liberalisation was decided upon with the idea of saving the lives of millions of women, who resorted to criminal abortion out of desperation, thereby succumbing to its complications.

As per this Act, pregnancy can be terminated on the following **grounds** :

Therapeutic: If the pregnancy, when allowed to continue, is likely to endanger the life of the woman, or cause serious injury to her physical or mental health.

Eugenic: If there is evidence to suggest that the child to be born would suffer from severe physical or mental abnormalities, for e.g., the mother has contracted any of the TORCH group of infections (toxoplasmosis, rubella, cytomegalovirus or herpes) in the first trimester.

Humanitarian: When pregnancy results from rape.

Social : If pregnancy has resulted due to contraceptive failure in a married woman, or if there are severe economic constraints in bringing up a child which can cause substantial burden on the family.

Qualifications Required: As per the Act, a medical officer is qualified to perform MTP if :

- He, as a registered medical practitioner has assisted in at least 25 cases of MTP in a recognised hospital.
- He is a specialist with MD (Gynaecology) or DGO qualification, or he has 6 months of experience in house-surgeonship in Gynaecology, in a recognised hospital.

Place: MTP can be carried out in any government hospital, or non-government institution, provided that a license has been procured from the Chief Medical Officer of the district.

Consent: Any woman over the age of 18 years can give written consent for an abortion. In the case of a minor or a lunatic, the consent of the guardian/ parent is required.

Duration of gestation: For a pregnancy of below 12 weeks duration, one medical officer alone can take the decision of performing an abortion, while if it is between 12-20 weeks, two medical officers are required to terminate the pregnancy. However if it is an emergency situation, a pregnancy exceeding 20 weeks can be terminated even if only one doctor is available.

COMMON METHODS OF MTP

- Dilatation and curettage
- Dilatation followed by oxytocin infusion
- Vacuum aspiration technique (VAT)
- Intraembryonic instillation of prostaglandin
- Extraembryonic instillation of hypertonic saline
- Hysterotomy

COMPLICATIONS[2]

Immediate complications include haemorrhagic shock, perforation of uterus, peritoneum or intestine, laceration of cervix or vagina, incomplete abortion, embolism, thrombophlebitis, etc. **Delayed complications** may take the form of pelvic inflammatory disease, menstrual irregularities, sterility, endometritis, cervicitis, vaginitis, etc.

CRIMINAL ABORTION

COMMON METHODS

When the pregnancy is in its initial stages, heavy physical exercise, hot baths, ingestion of purgatives, etc., are some of the methods advocated by criminal abortionists. When the pregnancy is in a more advanced stage, **abortifacient drugs** are favoured. These are conventionally (though obsoletely) classified into *ecbolics* and *emmenogogues*.

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Ecbolics stimulate uterine contractions and bring about abortion. Ergot preparations are usually used for this purpose. Lead oleate is also used, which is toxic to the foetus leading to its death. It may cause lead poisoning in the mother. Pituitary extract administered parenterally behaves in the same way as oxytocin.

Emmenogogues are drugs which promote uterine congestion and bleeding, resulting in the expulsion of products of conception. Examples include borax, oil of savin, rue, laburnum and sanguinarine.

Other **abortifacient drugs** (which act indirectly) include

Genito-urinary tract irritants such as cantharides, turpentine oil, etc., which act by producing inflammation of the genito-urinary tract, thereby reflexly irritating the uterus and stimulating its contractions.

Gastro-intestinal tract irritants such as tartar emetic, which can also cause reflex contractions of uterine muscle fibres.

Plant irritants such as seeds or unripe fruit of papaya and methi seeds (fenugreek). Calotropis is also commonly used.

Systemic poisons such as the salts of arsenic, lead, copper, antimony and mercury.

Mechanical Trauma: When drugs fail to bring about abortion, mechanical trauma is resorted to. This may take the form of primitive, and often barbaric methods of rupturing the membranes with the help of knitting needle, hairpin, stick, metal wire, coat hanger, screwdriver, etc. Once the membranes are ruptured, the products of conception get expelled.

Sometimes hot water, Savlon, or Dettol is injected under pressure, using a Higginson's syringe. Lysol, cresol, formalin or potassium permanganate may also be used. This process of injecting a fluid under pressure is called *syringing*. At times, instillation of hot water has resulted in death due to vagal inhibition, or due to embolism. A visit to the scene of the crime may reveal the presence of a syringe or remnants of the fluid used.

Some abortionists insert foreign bodies such as a stick, in order to dilate the cervix. More commonly, a laminaria tent is inserted into the cervix. Being hygroscopic in nature it absorbs water and swells up, thus dilating the cervix. This results in expulsion of the foetus. Other substances that have been used include sola pith, compressed sponge, bark of the slippery elm, etc.

Abortion stick: This is a method favoured by untrained midwives (*dais*) in the rural areas. A thin stick about 12 inches long, is wrapped in gauze or cotton wool. It is then dipped in an irritant solution like the juice of marking nut, or calotropis, or the paste of some metallic salt. The stick is then inserted into the cervix or vagina. It causes ulceration, bleeding from the os and expulsion of the foetus. At times, death of the woman takes place due to systemic absorption of the irritant or due to septicaemia. The application of potassium permanganate crystals can be extremely dangerous, causing ulceration, haemorrhage and toxic effects due to systemic absorption.[3]

Cupping: A lighted wick is placed over the hypogastrium and a cup is inverted over it. This creates a negative pressure, and when the cup is pulled suddenly, partial separation of placenta can result.[4] Other methods resorted to by criminal abortionists include introduction of pastes, curetting, application of electrical current locally, or insufflation of air.

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COMPLICATIONS OF CRIMINAL ABORTION

Immediate Complications:

- Haemorrhage due to perforation of vessels.
- Shock due to vagal inhibition resulting from instrumentation, or insertion of objects into the genital tract.
- Fat embolism may occur while syringing with soapy liquid.
- Air embolism due to insufflating air inadvertently into the uterine cavity during amateur procedures.

Delayed Complications:

Septicaemia.
Tetanus.
Endotoxic shock.
Renal failure
Meningitis, hepatitis, etc.

THE DUTIES OF A MEDICAL PRACTITIONER

When a lady comes to a doctor with history of incomplete abortion attempted by an abortionist or by herself, the medical practitioner must make a detailed note of the circumstances and the method employed to procure abortion. If death is imminent, he must record a dying declaration. If the patient dies, he must not issue a death certificate, but report to the Police and arrange for an autopsy to be done.

Medical Evidence of Abortion:

In a living victim

- The breasts show pigmentation.
- Secretion of milk or colostrum from the nipple.
- Linea nigra and/or albicans may be present.
- In advanced pregnancy, the uterus may be palpable even after abortion.
- Congestion of the labia majora and minora is often present.
- Tags of membrane may be found in the uterus.
- Ulceration or erosion of the vagina or cervix may be noted.
- A swab taken from the cervical canal may help in detecting the chemical used for syringing.
- Urine examination will reveal high levels of HCG up to 7 days.

In a dead victim (Autopsy Findings)

In addition to the above findings,

- The face may appear pale or bluish.
- Undergarments may show blood clots and fragments of products of conception.
- Examination of the genitalia often reveals congestion of the labia, and injury to the posterior commissure.
- The abdominal cavity may contain clotted blood.
- Perforation of pelvic organs may have occurred.
- Uterus may show the presence of paste, or marks of instrumentation.
- In the case of intrauterine infection, the uterus will be swollen, spongy, and discoloured. Serosal surface may be brownish, especially in clostridial infections. The endometrium may exhibit foul smelling purulent discharge.[5]

Medico-legal Importance of Placenta :

- Period of gestation can be made out by noting the extent of maturity of the placenta.
- Some poisons may be detected in the placenta.
- Retained placenta in criminal abortion may be the cause of death due to haemorrhage.

SECTIONS of the IPC DEALING WITH CRIMINAL ABORTION

312 : (Causing miscarriage)
Whoever voluntarily causes criminal abortion is liable for imprisonment upto 3 years and/or fine. The imprisonment may extend up to 7 years if the woman is quick with child. It is necessary that the woman should be pregnant and abortion carried out with her

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consent. Here the term 'with child' means pregnant, and 'quick' means the woman should have felt the child move within her.

313 : (Causing miscarriage without consent)
If miscarriage is carried out without the consent of the woman, the imprisonment may be extended up to 10 years.

314 : (Death caused by act done with intent to cause miscarriage)
If death of the woman takes place as a result of miscarriage, the minimum punishment would be 10 years.

315 : (Act done with intent to prevent the child being born alive, or to cause it to die after birth)
A person doing an act to prevent a child from being born alive, or causing it to die soon after it's birth is liable to punishment up to 10 years imprisonment.

316 : (Causing death of a quick, unborn child)
Causing death of a quick, unborn child amounts to culpable homicide, and the punishment may extend up to 10 years imprisonment.

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CHAPTER - 19

Infanticide

"But what am I? An infant crying in the night, An infant crying for the light, And with no language but a cry".

- Lord Alfred Tennyson

DEFINITIONS

Infanticide: Though the legal definition and implications of infanticide vary from country to country, the medical concept is more or less universally accepted. **Infanticide** refers to the *deliberate killing of a child below the age of one year.* In England, Germany, and the U.K, there is a clear-cut distinction between homicide and infanticide, and a charge of infanticide is less serious than a charge of murder, if committed by the mother due to stress factors surrounding birth and lactation.

In India, no such distinction exists, and the mother is punished under section 302 (IPC), the same as for murder.

Foeticide: Killing of a foetus prior to birth.

Neonaticide: Killing of a newborn infant. The perpetrator is invariably the mother, and the cause of death is usually either asphyxia or abandonment.[1]

Still Birth: Refers to the birth of a baby born after 28 weeks of gestation, which did not at any time after being completely expelled from its mother, breathe or show any other signs of life.

Dead Birth: A dead born child is one which has died in utero, and shows one of the following signs after it is completely born:

Maceration - This is a process of aseptic autolysis. Unlike putrefaction which is due to bacteria, there is no bacterial action in maceration. The cells break down due to enzymatic action (autolysis), and the body becomes soft and flaccid. The skin appears purplish brown and peels easily. Signs of maceration become evident only about 24 hours after the death of the foetus. However skin slippage can occur as early as 6 to 12 hours, and constitutes the earliest sign of maceration.[2]

Putrefaction - If there is rupture of the membranes, air entry is facilitated and putrefaction sets in.

Mummification - When liquor amnii is scanty, the foetus becomes dry and shriveled.

Rigor mortis - In the early stages after intra-uterine death, if the foetus has developed to nearly full term, rigor mortis may set in. The delivery of such a foetus may be difficult.

Spalding's sign - After the foetus dies in utero, the cerebral hemispheres shrink in size. As a result, the bones of the cranial vault loosen and begin to override each other. This overriding of the cranial vault seen on x-ray is suggestive of foetal death.

Viability: The physical ability of a foetus to lead a separate existence after birth, by virtue of a certain

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degree of development, is called **viability**. Although in some cases, infants have reportedly survived even though the gestation period was only 180 days, for practical purposes, the minimum duration required to make a foetus viable is taken as 210 days (7 calender months).*

Some factors which help in assessing the viability include *weight*, *crown-heel length*, and appearance of *ossification centers*. At seven months, the average weight is 1.3 kg, and the crown-heel length is 35 cm.

Rule of Haase - It is a rough method of determining the gestational age of a foetus after delivery. The square root of the crown-heel length (cm) gives the gestational age (months), up to 5 months intrauterine life. For e.g., a length of 16 cm indicates the age of the foetus to be 4 months. Beyond the 5th month, the length of the foetus (cm) when divided by 5 provides its age in months. For e.g., a length of 30 cm divided by 5 shows the age to be 6 months.

Ossification centers - By the 28th week, the centre for the proximal end of the tibia, as well as the centres for cuneate, capitate, and cuboid bones appear. The centre for the proximal end of the tibia is the most reliable, that of the cuneate and capitate least reliable, and that of the cuboid is of intermediate value.[3]

Live Birth: The term **live birth** is used when a child has showed signs of life (however slight it might have been), even if only a part of the child was out of the womb, though the child may not have breathed or been born completely. Causing the death of such a child is treated in the same manner as homicide.[4]

Signs of live birth - In India, the law presumes that every child that was discovered in a dead condition was born dead, unless the contrary can be proved. In civil cases, any sign of life such as a cry, movement of the body, or hand or foot, after birth is likely to be accepted as proof of live birth. Sometimes a child may cry even while it is still in the uterus or vagina (after the membranes have ruptured). This is called *vagitus uterinus* and *vagitus vaginalis*, respectively. And yet when the child is actually born, i.e., has come to the exterior, it may be found to be without signs of life. Hence, in criminal cases, the cry of the child alone is not proof enough of live birth. Such proof that is acceptable to the court can only be furnished after postmortem examination.

- *Umbilical cord*: If the cord has separated, it indicates that birth took place several days earlier. Generally, in the first 12-24 hours after birth, the umbilical cord becomes dry and shriveled. On the second day, a zone of redness is seen at the attachment of the cord to the umbilicus. It is also important to consider as to whether the cord was cut or torn. In the case of a torn cord, the edges appear ragged and ripped.

- *Presence of vernix*: This merely indicates that the child was not washed. It does not indicate live birth.

- *Food in the stomach*: Presence of milk or food in the stomach is a conclusive sign of live birth.

- *Proof of breathing*: The following points are relevant -
  - The shape of the chest is flat before respiration, after which it becomes drum shaped.
  - The diaphragm is at the level of the 6th or 7th rib after respiration has taken place. Otherwise it is at the level of the 3rd or 4th rib.

*Each calender month corresponds to approximately 30 days, while a **lunar month** is taken to be 28 days. Obstetric calculations are generally in terms of lunar months.

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- The lungs are voluminous, with the medial edges overlapping the mediastinum. The margins become rounded after respiration is established. Before respiration the lungs are firm in consistency, but respired lungs are spongy, elastic and crepitant, due to being distended with air.

- *Hydrostatic test*: The hydrostatic test is based on the principle that if the infant has breathed, the lungs will float in water. Each lung is cut into pieces and tested for floatation in water. A piece of liver can serve as control. If the liver bit floats, the test has no meaning, because it is clear that putrefaction has set in. If all the pieces of lung float, they are crushed under a weight and tested again.

  Inference: Floatation is positive when the baby has respired. But if the pieces sink after pressure, no respiration has taken place. If some pieces float while others sink, it means feeble respiration has taken place.

  Drawbacks: Expanded lungs may sink due to oedema, pneumonia, atelectasis, and obstruction by alveolar duct membrane. On the other hand, unexpanded lungs may float due to putrefaction or artificial respiration.

  The hydrostatic test is superfluous if the foetus is:

  below 180 days of gestation,
  macerated,
  a monster,
  showing milk in the stomach.

  The test is of limited value. It can only be used as a suggestive pointer to live birth, but it is not conclusive.

- Other indicators:
  - The lungs of a stillborn are dark, small and heavy (like liver).
  - Before respiration, the lungs are smaller, constituting about 1/70th of the body weight. After respiration, due to increased vascular volume it becomes 1/35th of the body weight (this is also sometimes called the **Ploucquet's test**).
  - The texture of the unrespired lung is rubbery and firm with sharp margins. On slicing, the interior is uniform in colour and texture. On rubbing a small piece between the fingers, close to the ears, no sound is heard.[5] In the case of a respired lung, crepitation can be made out.
  - Histological appearance: In a respired lung, alveoli appear expanded with flattened epithelium.
  - Presence of air in the stomach and intestine (which float in water if double-ligated). This is referred to as **Breslau's second life test** or **Stomach-bowel test**.
  - The replacement of gelatinous tissue in the middle ear after respiration is established may point to maturity of the infant (**Wredin's test**).
  - Foetal hemoglobin, which is 80% at birth, decreases to 7-8% in 3 months.

CAUSES OF INFANT DEATH

Death of a foetus may be due to natural or unnatural causes. **Natural causes** include immaturity, foetal malformations, and birth trauma. **Unnatural causes** may be accidental or criminal. *Accidental causes* may be due to prolonged labour, cord twisting around the neck, or cord prolapse. Sometimes, a foetus is born with the membranes covering the head (caul) which if not incised soon after birth, may lead to asphyxia.

Precipitate labour - Precipitate labour is said to occur when all the stages of labour merge with each other, and the birth takes place in an abnormally short time. The mother may experience mild pain

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which she may attribute to a distended rectum, and therefore she may attempt to defecate, as a result of which the child may be born into the lavatory pan. The child may sustain skull fractures or bleed from the torn end of the umbilicus. It may inhale faecal matter which may be demonstrable in the lungs.

Precipitate labour occurs when the pelvis is very roomy and the foetus is very small. In such cases, caput succedaneum or cephalhaematoma which are pointers to obstructed labour, are absent. Caput succedaneum forms in the scalp at the presenting part of the head in vertex presentations. The scalp is apposed against the cervix and congestion and oedema develop. It disappears within a week. Cephalhaematoma is due to accumulation of blood deep to the scalp, between the periosteum and the bone. It can persist up to 2 months.

Criminal causes of infant death constitute the offence of **infanticide**. These may be the result of acts of omission or acts of commission.

Acts of omission (or neglect) : Failure to
- protect the child from heat or cold,
- provide assistance during labour,
- clear the air passages of mucus,
- cut the umbilical cord,
- feed the child.

Acts of commission : The following constitute some common examples -
- Smothering, e.g., by blocking the air passage by a pillow or soft cloth.
- Strangulation, e.g., by ligature, sash of the baby's frock, or even the umbilical cord.
- Manual strangulation or throttling.
- Banging the head of the child against a wall.
- Burning.
- Drowning.
- Concealed puncture wounds, e.g., in the axilla, inner canthus of the eye, vagina, rectum, through a fontanelle, etc., usually accomplished by the insertion of a stout needle or fine-bladed knife.
- Poisoning.

CONCEALMENT OF BIRTH

Section 318 of the Indian Penal Code recognizes intentional concealment of birth and/or secret burial or disposal of a dead child as a crime punishable with fine or imprisonment up to 2 years, irrespective of whether the child dies during, before, or after birth.

ABANDONING OF INFANTS

If the parent or guardian of a child below 12 years, or anyone entrusted with the care of such a child, leaves it in any place with the intention of abandonment, he /she is punishable up to 7 years imprisonment (sec 317 IPC)

BATTERED BABY SYNDROME

Synonyms

Non-accidental Injury of Childhood, Caffey's Syndrome, Child Abuse Syndrome, and Maltreatment Syndrome

Definition

A battered child is one that suffers repetitive physical injuries inflicted by a parent or guardian, which are non-accidental in nature.

Manifestations

The signs to look for in a suspected victim of battering include :
- Bruises around the wrist, forearm, thighs, and ankles, which form 'handles' for adults to grip and swing the child.
- The buttocks may show burns, or whip marks
- The face and lips may be bruised. The frenulum of the tongue may be torn.

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- Circular bruises of 1-2 cm size, called 'six-penny bruises' which occur due to the poking of adult fingers on chest, abdomen and thighs.[6]
- Bruises of different periodicity on the same child with a history inconsistent with the findings.
- Crack fractures of the skull bones associated with intracranial hemorrhages.
- Multiple rib fractures may be seen.
- Limb fractures are mostly in the region of the metaphysis and epiphysis of growing bones. Periosteal elevations may be seen.

Cinderella Syndrome – Sometimes in a family, a single child is chosen to receive the battering. The child (commonly the youngest or eldest) is repeatedly thrashed, while the other children of the same family are spared.

SUDDEN INFANT DEATH SYNDROME

Synonyms

Cot death, Crib death, SIDS.

Definition

SIDS is defined as *the sudden death of an infant which remains unexplained after a thorough case investigation, including performance of a complete autopsy, examination of the death scene, and review of the clinical history.*[7]

The incidence of SIDS is 2-3/1000 live births, with a slight male preponderance. Most cases involve infants between the ages of 2 weeks to 2 years, with maximum incidence between 3 and 7 months. Crib deaths occur commonly at night or in the wee hours of the morning. Twins are at greater risk. In many cases, there is a prior history of running nose or coryza.

Autopsy findings

- Blood stained froth in the mouth.
- Evidence of laryngitis, tracheitis, tracheobronchitis, or congenital heart disease may be seen in some cases.
- Multiple petechial haemorrhages are often noticed in the heart, lungs, and thymus.

Aetiology

The hypotheses for Sudden Infant Death Syndrome include:

- Overlaying of the infant by drunken parents.
- Prone sleeping position.[8]
- Inadvertent smothering by soft cloth or pillow.
- Sleep apnoea, which causes hypoxia and respiratory depression.
- Hypotonic babies, whose neck posture reduces the airway lumen.
- Dust mite allergy.
- Cow's milk allergy.
- Calcium or selenium deficiency.
- Viraemia.
- Hyperthermia or hypothermia.
- Anaphylactic shock.

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CHAPTER - 20

Sexual Offences and Paraphilias

"Sometimes when I'am doing it with a woman, I get this urge to hurt her, to draw her blood. I just can't help it....".

- **Ted Bundy** (Serial rapist-murderer)

Given the conduct of civilized society, both law and custom permit only heterosexual natural sexual intercourse (i.e., penile-vaginal sexual intercourse) between man and wife. A **sexual offence** is therefore defined as *any form of sexual intercourse which departs from this norm, and is consequently contrary to law.*

Sexual offences are classified as:

A. Natural sexual offences
1. Rape
2. Adultery
3. Incest

B. Unnatural sexual offences
1. Sodomy
2. Bestiality
3. Buccal coitus
4. Lesbianism

C. Sexual deviations/perversions/paraphilias
1. Fetichism/transvestism
2. Sexual sadism/masochism
3. Exhibitionism/voyeurism
4. Paedophilia
5. Miscellaneous disorders: necrophilia, necrophagia, etc.

NATURAL SEXUAL OFFENCES

RAPE

Rape is defined in **Sec. 375, IPC**[1] as *unlawful sexual intercourse by a man with a woman*
a. against her will, or
b. without her consent, or
c. with her consent,when her consent is obtained by
   i. force, or
   ii. putting her or any other person in whom she is interested, in fear of bodily hurt or death, or
   iii. when he knows that he is not her husband, and that her consent has been given because she believes that he is the man to whom she is lawfully wedded; or
d. with or without her consent
   i. when she is of unsound mind, whether due to mental disease or defect, or intoxication, or
   ii. when she is under16 years of age.

Sec. 375, IPC also explains that mere penetration is sufficient to constitute the offence of rape, and provides an **exception** to the age of valid consent to sexual intercourse by a female, i.e., *sexual intercourse by a man with his own wife, the wife*

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being not less than fifteen year of age, is not rape. Further, a **Manipur state amendment** to rape law has reduced the ages of valid consent to sexual intercourse by unmarried and married females to 14 and 13 years respectively.

From the **psychological viewpoint,** it has been said that "*man's discovery that his genitalia could serve as a weapon to generate fear must rank as one of the most important discoveries of prehistoric times*", and, "*rape is one way in many (but not all) societies that men as a class oppress and control women as a class.*"[2] Therefore, it is obvious that rape is as old as human society, and that it is a process of intimidation of women, and dispossession of what the victims (and their patriarchal husbands, classes and societies) consider as their most valuable asset.

It must be emphasized here that **rape is not a medically recognized entity, but a sociological and legal concept.**

Before proceeding any further, it would be pertinent to critically analyse from a medical viewpoint the provisions of **Sec. 375, IPC**:

Firstly, to constitute rape, sexual intercourse must be **unlawful,** i.e., between two persons who are not lawfully married. (Please note that coitus between a man and a woman who are not lawfully married to each other, but both also being unmarried, is not "unlawful" as long as such coitus takes place by mutual consent. Such sexual intercourse is, however, considered by orthodox societies as **illicit,** i.e., not permitted,[3] and is called by them "fornication"). However, coitus between man and wife also amounts to rape if the wife is less than 15 years of age, because Indian law considers that, in view of her tender age and consequent immaturity of mind, the act may affect her body and mind adversely; hence the husband has no absolute right to coitus with her. (It may be mentioned here that the age of valid consent to sexual intercourse by a married woman as per Sec.375, IPC is itself a contradiction, as both the **Child Marriage Restraint Act** and the **Hindu Marriage Act** clearly specify that the minimum age at which a female can validly enter into a contract of marriage is eighteen years!)

Secondly, a **man** is defined in the Indian Penal Code as *a male human being of any age.* Therefore, there is, as per Indian law, no age below which a "man" cannot be charged with the offence of rape, unless Sec. 82 and 83, IPC can be applied, the latter stating that "Nothing is an offence which is done by a child above seven years of age and under twelve, who has not attained sufficient maturity of understanding to judge the nature and consequences of his conduct."

The question of *minimum age* of the woman does not arise, because victims of rape have ranged from infants to the old and infirm.

Thirdly, there is bound to be some confusion between "will" and "consent" because in the minds of most people, the two words are synonymous. The two, however, differ considerably in that "will", in this context, is most simply explained as *desire, wish, disposition, choice or determination,* whereas "consent" is defined as *compliance in or approval of what is done or proposed by another.* Therefore, a person may be willing to do something, but may not agree to do it. On the other hand, one may not be willing to do something, yet may comply, for whatever reason, with what is demanded. In both cases, it is most often when a person has no choice in the matter that either will, or consent, or both are suborned. (With regard to consent, some of the conditions under which consent is invalid are enumerated in Sec. 90, IPC.) It is important to note that legally valid consent by a woman to sexual intercourse, whether implied or expressed, **must be given before the act – that obtained subsequent to the act is not acceptable to a court of law.**

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Indian law also requires that unwillingness or lack of consent by the woman be proven by signs of resistance on her person, such as damage to her clothing or injuries on her body. This could be possible only if the victim was in a position to put up a fight; it would be futile to search for such evidence if the victim had changed her clothing after the incident (or it was destroyed), or was unable to (or could not) resist because of fear, fraud, impersonation, temporary or permanent unsoundness of mind, or lack of knowledge of the nature and consequences of the act.

Fourthly, Sec. 375 explains that **mere penetration** (even up to the vulva) is sufficient to constitute the offence of rape – neither injury to the external genitalia, nor rupture of the hymen, nor penetration into the vagina, nor seminal emission is essential. Similarly, the inability to achieve or sustain erection does not preclude the inability to commit rape, and the presence of abrasions, and absence of a uniform layer of smegma over the glans penis indicate only complete sexual intercourse, not rape.

All the above factors considered, the offence of rape can be committed without any trace left on either victim or perpetrator. Any medical evidence found could as easily be located in circumstances that do not constitute rape. Conversely, its absence does not necessarily mean that an alleged victim has not been raped.

Finally, an interesting point of argument is "*Can a woman rape a man, or a man rape a man, or a woman rape a woman?*" In any of these contexts, although the word "rape" may be inappropriate, it must be admitted that sexual intercourse, whether natural or unnatural, can be performed against the will and/or without the legally valid consent of the other party. The issue has been somewhat resolved by the **Florida Sexual Battery Bill** [4] which has repealed the former rape law in that state, and defines **sexual battery** as *oral, anal or vaginal penetration by, or union with the sexual organs of another, or the anal or vaginal penetration of another, by any other object.*

The **punishment** for **rape** is prescribed in **Sec. 376, IPC**

a) **Subsection 1 :** For rape as described in Sec. 375, imprisonment for at least seven years (which may extend to ten) and liability to fine, unless the victim is his own wife and is not less than twelve years of age, in which case the maximum sentence is two years of imprisonment, or fine, or both.

b) **Subsection 2 :** Deals with **custodial rape** (that committed by a police officer or a public servant on a woman in prescribed situations), **institutional rape** (by a person on the management or staff of places such as remand homes, jails, women's or children's institutions, hospitals, etc.), rape of a **pregnant woman** or a **woman less than twelve years of age,** or **a victim of gang rape** (that perpetrated by "one or more of a group of persons acting in furtherance of a common intention."). All of these being considered by both law and society as more heinous offences than rape under Sec. 375, IPC, because of either the criminal abuse of authority, or the vulnerability of the victim, the minimum duration of the penalty is enhanced to ten years. **Box 20.1** highlights the landmark case which led to **The Criminal Law (Amendment) Act, 1983,** which in turn led to the inclusion of the specific offence of custodial rape, as well as enhancement of minimum punishment for the offence of rape in general.

c) Further, under
   i) **Sec. 376-A,** *a husband who "has sexual intercourse with his own wife, who is living separately"* while divorce proceedings are

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Box 20.1 The Mathura Case (1972) (*Tukaram Vs State of Maharashtra*)

Mathura, a 15 year old Harijan orphan girl was called to the Desaigunj police station of Chandrapur district of Maharashtra, on an abduction report filed by her brother, Gama. Mathura had attempted to elope with a neighbourhood boy, Ashok, since she had fallen in love with him. During the course of investigation of the case, Mathura, along with others, was detained in police custody. When it was time for them to leave however, Mathura was asked to stay back at the police station by two constables on duty at that time : Ganpat and Tukaram. It was late at night, and there was nobody else at the police station. Ganpat forced Mathura into a toilet and raped her. Tukaram followed suit, but owing to intoxicated state did not actually accomplish the act, though he fondled and molested the helpless girl.

The next morning, Mathura narrated her harrowing tale to her brother who reported the matter to senior police officials. The two constables were arrested and a case of rape was filed against them. On trial, the Sessions Judge of Chandrapur exonerated the culprits. He held that the constable Ganpat was not guilty of rape, though he accepted the probability of sexual intercourse between Ganpat and Mathura at the police station. According to him, there was a world of difference between *sexual intercourse* and *rape.* In the present case, he was of the opinion that the offence of rape had not been proved.

On appeal, the Bombay High Court (Nagpur Branch), reversed the finding of the Sessions Judge, and found Ganpat guilty of rape and sentenced him to 5 years rigorous imprisonment. Tukaram was sentenced to 1 year imprisonment for molesting the girl.

Against the judgement of the Bombay High Court, the accused appealed to the Supreme Court which, on September 15, 1978, shockingly reversed the finding of the High Court. It held among other reasons, that only "fear of death or hurt" can vitiate consent for sexual intercourse, and no such finding was recorded. The court further observed that Mathura was not subjected to any fear, and that she had in all probability voluntarily consented to the act. The two constables were acquitted of all charges.

The judgement of the Supreme Court led to vociferous debate among sections of the general public. Women's Rights Groups widely criticised the judgement, and stated that it involved sacrificing human rights of women under the law and the Constitution. Mathura was after all a young, illiterate, orphan girl from a backward community, and she was all alone in the dead of night in a police station, with two burly men who were supposed to uphold the law, and protect her. How could anyone expect her to put up stiff resistance in such circumstances? Public outcry led to a review petition being filed by the Maharashtra Government, but the Supreme Court dismissed it on an unrelated technical point, without going into the merits of the case.

The controversy generated by the Mathura case however served to change the existing rape law, and led to the enactment of a more stringent law (**Criminal Law Amendment Act, 1983**), which introduced the provision of a new offence (*custodial rape*), and enhanced the minimum punishment for all categories of rape.

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pending in court, can be punished with a maximum of two years of imprisonment, and

ii) **Sec. 376-B,C & D,** *a public servant, superintendent or member of the* management or staff of any of the institutions previously listed, who has sexual intercourse with any inmate of such an institution, such sexual intercourse not amounting to *rape,* can be punished with imprisonment for a maximum duration of five years.

MEDICOLEGAL EXAMINATION OF AN ALLEGED VICTIM OF RAPE[5]

A physician's responsibilities in the medicolegal examination of the victim of an alleged sexual assault are both *therapeutic*, in ameliorating the physical and emotional consequences of the incident, as well as *evidentiary*, in order to facilitate the scientific investigation of the crime. Thus, medical and paramedical staff need to be aware of the definitions of, and common misconceptions and attitudes concerning rape, and familiarise themselves with the procedures for collection and despatch of material evidence.

Written informed consent of the victim to examination and treatment, as well as for the collection of material evidence, and the subsequent release to legal authorities of information obtained during the examination, duly **witnessed,** must be obtained.

A female nurse must be present throughout, not only to assist the examining physician (who must preferably be a female), but also to preempt allegations of sexual assault against the physician (if this happens to be a male), and to provide emotional support to the victim.

The medicolegal examination should include complete **history, physical examination, and collection of material evidence** for laboratory examination, to establish the following crucial points :

1. Did sexual intercourse occur?
2. If yes, was this sexual intercourse forced on the alleged victim?
3. Was the alleged victim a virgin prior to the commission of the alleged offence?
4. If the validity of consent is the question, a) how old is the victim? b) was she of sound or unsound mind (whether due to mental disease or defect, or intoxication) at the time of commission of the alleged offence?
5. How many assailants were there?
6. Is it possible to identify the assailant/assailants from material evidence gathered?
7. Are there any identifiable sequelae of the alleged offence (e.g., sexually transmitted disease, pregnancy or psychological trauma)?
8. How much time has elapsed between commission of the alleged offence and medicolegal examination?

THE HISTORY

The purpose of the history (which includes a general medical history, a sexual history, and a history of the incident and post-incident events) is to gather information that will help the medical and legal management of the case.

i) **The general medical history** should include:
- a past medical history of injury or illness (particularly disorders which may have an adverse effect on the alleged victim, e.g., bleeding disorders).
- any medications or allergies to medications.
- past immunizations.

ii) Points to be noted in the **sexual history** are
- Has the victim attained menarche ?
- If yes, what is the frequency and regularity of menstruation?
- Has the victim been sterilized, or does she use any contraceptives?

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- Marital and obstetric history (where applicable):
  - Number of pregnancies, and the result of each.
  - Most recent consensual sexual intercourse.
  - Existence and onset of any symptoms and/or signs of pregnancy.
  - Patient's attitude to pregnancy and its prevention or termination by mechanical or chemotherapeutic measures.
- Past history of venereal disease:
- Whether treated or not, and if yes, where and when, and whether blood titres returned to normal.

iii) **A history of the incident, and post-incident events** should include statements regarding
- time and location of the alleged assault,
- specific nature of the alleged assault,
- whether penetration was vaginal, oral, or anal,
- whether or not body fluids (through ejaculation, oral manipulation, urination, or defecation) were left in/on the victim,
- whether or not instruments and/or restraints were used,
- number of assailants, and
- whether or not there were any activities which could possibly interfere with the collection of material evidence (change of clothing, bathing, douching, urinating, defecating, etc).

It must be emphasized here that *the history should be taken with care in a sensitive way, using the patient's own language and in terms familiar to her.*

THE EXAMINATION

It must be conducted gently, but firmly, in the presence of a female nurse, explained step by step after ensuring acoustic and visual privacy, and thorough enough to note any abnormalities resulting from the sexual assault.

i) **General examination:** (Fig 20.1)
- Take the victim's (or that of the parent/guardian) written informed consent.
- Assess and record the victim's general appearance (disarrangement or damage to, and/or any stains on clothing, demeanour, signs of emotional distress, and gait).
- Record vital parameters (blood pressure, pulse, temperature and respiration).
- Record at least two marks of identification.
- Request the victim to stand on a large clean white sheet of paper, and to undress herself, and collect any material that falls onto the paper in the process.
- Examine the fingernails for any damage, and for any material which may have accumulated under them, which must be scraped off into cellophane packets (to be sealed, labeled and forwarded to the Forensic Science Laboratory for examination).
- Record the presence of fresh/dried stains of blood, semen, dirt, mud, grass, etc., on the surface of the body ; these too, must be swabbed/scraped off into appropriate containers, sealed, labeled, and despatched to the Forensic Science Laboratory.
- Carefully examine and meticulously record external injuries (signs of restraint, bite marks, scratches, abrasions with/without contusions, etc.).

ii) **Local (genital) examination** (Fig 20.2), should be directed towards:
- the external genitalia and its surroundings:
  - pubic hair : matted with blood/semen? combings for specimens of foreign hair. clippings for comparison.
  - bruising of the inner aspects of the thighs and labia.

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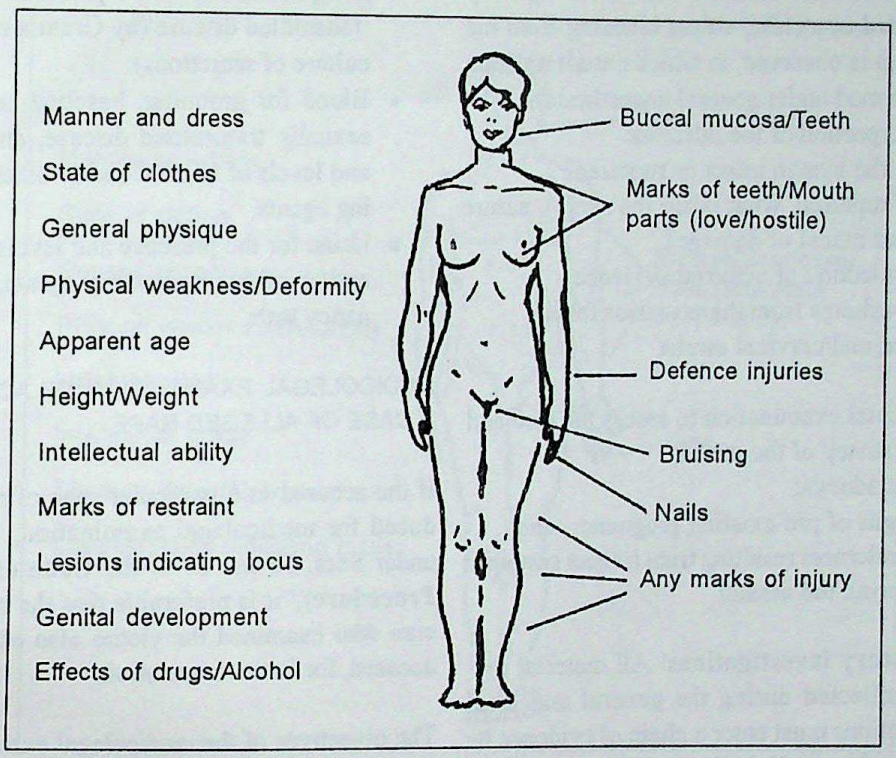

Fig. 20.1 General examination of rape victim

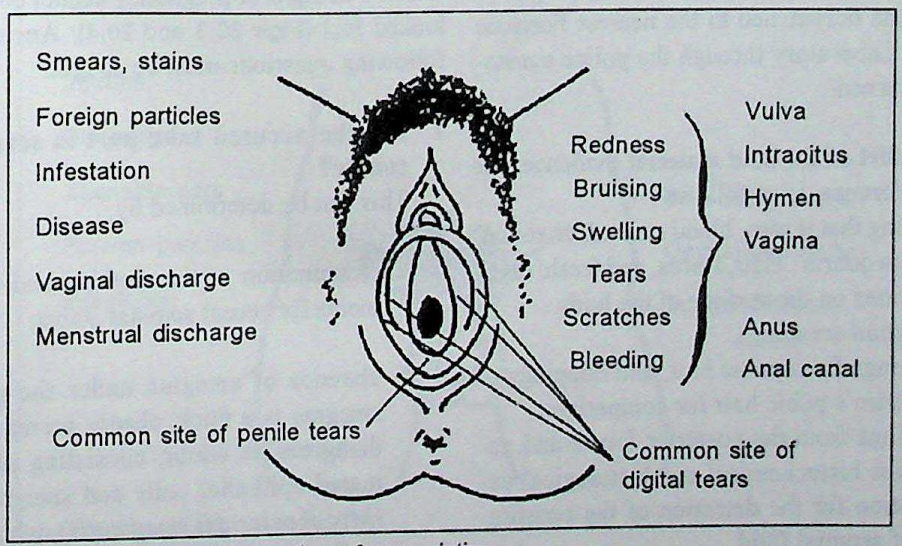

Fig. 20.2 Local examination of rape victim

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- internal examination (which is usually avoided in a child, unless bleeding from the vagina is observed, in which case it must be performed under general anaesthesia):
  - inspection of the introitus:
    is the hymen intact or ruptured?
    if ruptured, what is/are the site(s), nature and extent of damage?
  - collection of material evidence :
    washings from the posterior fornix, vaginal/cervical swabs.

- bimanual examination to assess the size and consistency of the uterus,
  - its adnexa;
  - signs of pre-existing pregnancy, and
  - tenderness resulting from trauma sustained during the assault.

iii) **Laboratory investigations**: All material evidence collected during the general and local examinations must enter a chain of evidence by proper sealing and labeling, with the name, case record number, date and time of collection, and the name and designation of the examining physician, and despatched to the nearest Forensic Science Laboratory through the police constable concerned.

A checklist of the said material evidence, for ready reference, is as follows :

- Clothing that is torn, blood stained, or soiled.
- Foreign debris : Hair, fibres, and fresh/ dried secretions on the surface of the body.
- Fingernail scrapings.
- Combings from pubic hair, and clippings of the victim's pubic hair for comparison.
- Washings from the posterior fornix and vagina, for histochemical and histological examination for the detection of the constituents of seminal fluid.
- Vaginal and cervical swabs for the detection of the above, and of *Mycobacterium smegmatis*, and for the presence of sexually transmitted disease (by Gram's staining and culture of secretions).
- Blood for grouping, baseline serology for sexually transmitted disease, and presence and levels of ethanol and/or other intoxicating agents.
- Urine for the presence and levels of ethanol and/or other intoxicating agents, and pregnancy tests.

MEDICOLEGAL EXAMINATION OF ACCUSED IN A CASE OF ALLEGED RAPE

If the accused in a particular case of rape is produced for medicolegal examination, (conducted under **Secs. 53** and **54** of the **Code of Criminal Procedure**),[6] it is preferable that the same physician who examined the victim also examines the accused, for better correlation.

The objectives of the medicolegal examination do not differ significantly from that of the examination of the victim, except that such sequelae of the incident as signs of pregnancy cannot obviously be looked for! (**Figs 20.3** and **20.4**). Answers to the following questions must be sought:

1. **Did the accused take part in sexual intercourse?**
   This can be determined by:

   a. examination of the clothing and undergarments for recent seminal stains;

   b. absence of smegma under the prepuce : smegma is a thick, cheesy secretion with a disagreeable odour, consisting of desquamated epithelial cells and smegma bacilli (*Mycobacterium smegmatis*), which is generally wiped off during sexual intercourse. However, its absence is not conclusive evidence of sexual intercourse, since during the

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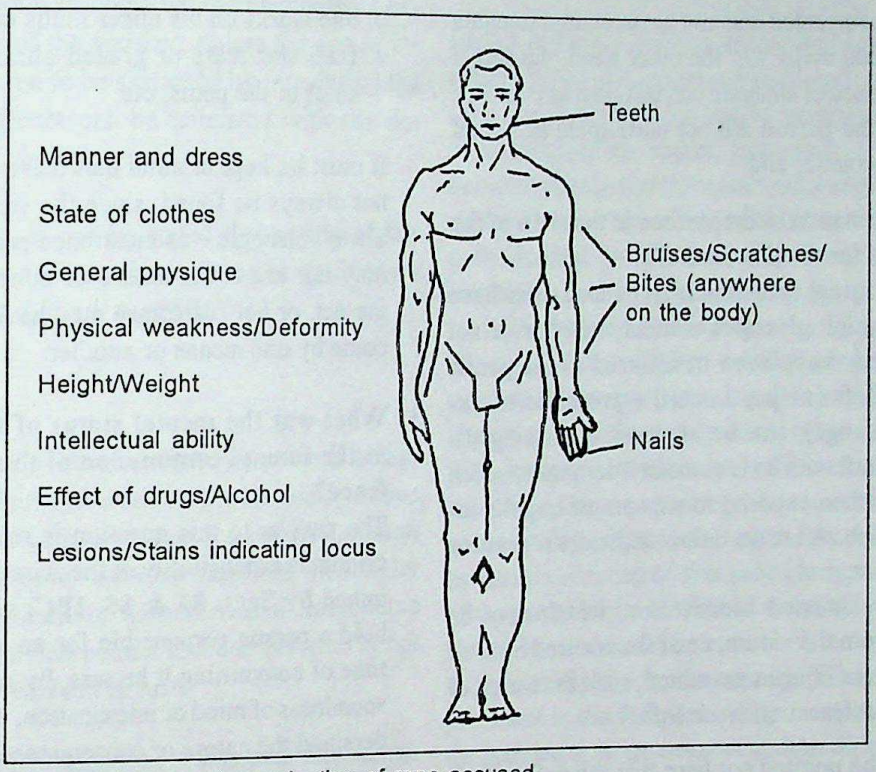

Fig. 20.3 General examination of rape accused

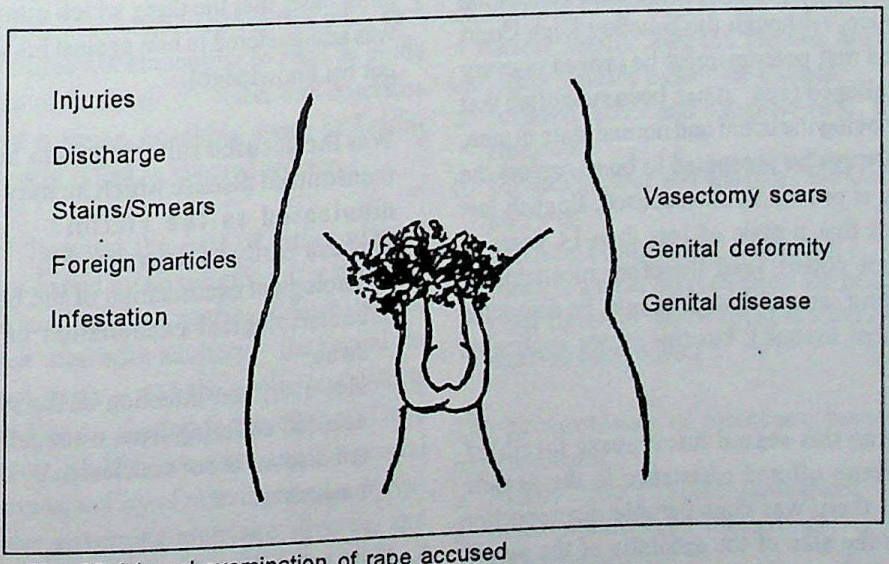

Fig. 20.4 Local examination of rape accused

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customary daily bath, the prepuce is expected to be retracted and any accumulated smegma washed away. On the other hand, the actual presence of smegma can be taken as evidence that the person did not participate in sexual intercourse; and

c. examination of the surface of the shaft of the penis for the presence of
   i) vaginal epithelial cells : Since these have a high glycogen content, whether or not they have been transferred to the penile shaft (as per Locard's principle of exchange), can be assessed by wiping the shaft with a clean moist filter paper, which is then exposed to vapours of Lugol's iodine. A brown colour indicates a positive test; and
   ii) fresh/dried bloodstains, which may be from the victim, or of the accused himself from injuries sustained, such as tearing of the fraenum, (*vide infra*).

It must be pointed out here that the defence often raises the plea that the accused was impotent, and , therefore, could not have committed the offence. Although the Bombay High Court had ruled that potency must be proved in every case of alleged rape, it has been submitted that potency being the usual and normal state in man, it will always be presumed to be so, unless the contrary is proved by the accused. English law presumes that a male of less than 14 years of age is not potent, (and therefore incapable of committing rape, although he may be convicted of indecent assault), but this is not applicable in India.

2. **If yes, was this sexual intercourse forcible?**
If the victim offered resistance to the assault, and/or if there was considerable disproportion between the size of the genitalia of the assailant and victim, the assailant may have sustained injuries such as:
   a. scratches on the face;
   b. bite marks on his upper limbs or elsewhere;
   c. fraenular tears or grazed abrasions on the shaft of the penis, etc.

It must be kept in mind that these injuries will not always be found, since the victim will not always struggle – as mentioned previously, she may not know the nature or consequences of the act, or her resistance may have been overcome by one means or another.

3. **What was the mental status of the accused at the time of commission of the alleged offence?**
The answer to this question is relevant to the criminal responsibility of the accused, as determined by **Secs. 84 & 85, IPC,** which do not hold a person responsible for an act if, at the time of committing it he was, by reason of unsoundness of mind or intoxication, unable to understand the nature or consequences of what he was doing, or that he was doing what was either wrong or contrary to law (provided, in the latter case, that the thing which intoxicated him was administered to him against his will or without his knowledge).

4. **Was the accused suffering from any sexually transmitted disease which he may have communicated to the victim?**
This can easily be determined by
   a. serological examination of the blood, and
   b. bacteriological examination of a urethral swab.

   However, non-infection of the victim by an accused suffering from a sexually transmitted disease is not conclusive evidence of his innocence.

5. **How much time has elapsed between commission of the alleged offence and medicolegal examination?**

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This is probably the most difficult to answer of the questions arising from the medicolegal examination of the accused, unless the age of injuries proven to be sustained by him during the alleged offence can be estimated with any degree of accuracy.

In conclusion, it can be stated that courts of law consider rape as "..... *the easiest charge to make and the most difficult to refute.*"

ADULTERY

Adultery is defined as *voluntary (natural) sexual intercourse between a married man and someone other than his wife, or between a married woman and someone other than her husband.* In simpler terms, it is voluntary natural sexual intercourse between a married person and another who is not one's legally wedded spouse.

Sec. 497, IPC, states that, "Whoever has sexual intercourse with a person whom he knows or has reason to believe to be the wife of another man, without the consent or connivance of that man, such sexual intercourse not amounting to rape, is guilty of the offence of adultery....." **Sec. 498** describes the offence of enticing, or taking away, or detaining with criminal intent, a married woman.

Although adultery on the part of either man or woman is proscribed by all religions, it is odd that, under Indian law, only an aggrieved husband can charge another man with adultery – the sexual reverse does not apply – and the adulterous wife is not considered as an abettor to the offence. Once proven, however, adultery constitutes a valid ground for divorce and/or nullity of marriage under prevalent civil codes governing marriage, divorce, and nullity of marriage in our country.

INCEST

Incest is simply defined as *natural sexual intercourse between two persons so closely related that they are forbidden by law to marry.* Or, from a psychological viewpoint, *heterosexual relations between persons of the same culturally or legally defined group.* The extent of this "culturally or legally defined group" does, of course, differ from country to country, so that incest is punishable by legislation and constitutes a valid ground for divorce,and is prohibited by religious laws (by annulment of marriage) in many developed countries. However, in India and in many other Asian countries, incest is not a criminal offence (unless it also amounts to rape or adultery) because of the social acceptability of intracaste marriage. Although the ostensible purpose of this practice is maintenance of the purity of the bloodline, the pragmatic reason would appear to be retention of material assets within the family or community. What is not appreciated is the fact that such a custom has led to the geometric progression of genetic defects in such societies.

Incest most frequently occurs between members of a nuclear family, and the victims are most often children. Notable examples include

a. between father and daughter (e.g., the *Electra complex*),
b. between mother and son (e.g., the *Oedipus complex*), or
c. between brother and sister (e.g., "*Pharaonic incest*", a term derived from the practice in ancient Egypt in which royal brothers and sisters married each other).

The circumstances of incest are both social and environmental, i.e.,

a. Family strife and disorganisation,
b. In lower socioeconomic groups:
   i. Overcrowding,

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ii. Lack of parental supervision, and
iii. Low morality and delinquency.

c. In both a. and b. above, the psychological basis is:
   i. a fundamental psychological conflict in infancy and adolescence and/or
   ii. the repression of a forbidden desire for a parent or sibling.

UNNATURAL SEXUAL OFFENCES

As previously mentioned, the law in India to date permits only natural sexual intercourse between a legally married man and wife. **Sec. 377, IPC** states that *"Whoever voluntarily has carnal intercourse against the order of nature with any man, woman, or animal shall be punished with imprisonment for life, or with imprisonment of either description for a term which may extend to ten years, and shall also be liable to fine."* As with rape, penetration is sufficient to constitute the carnal intercourse necessary to the offence.

SODOMY

Sodomy is a type of unnatural sexual offence which derives its name from a historical city mentioned in the Bible, where it was supposed to have been practiced,[8] (although the Bible does not specifically describe it); it denotes *homosexual penile-anal intercourse. Heterosexual penile-anal intercourse* is called **buggery**.

The offender is called the *active agent*, while the other partner (who may or may not have consented to the act) is termed the *passive agent*. If the passive agent is a child, the practice is known as **paederasty**. Habitual passive agents are called *catamites* (also *fairies*, *gays*, or *queens*, in the West); in our country, *hijras* (castrated males), and *zenanas* (male transvestites) prostitute themselves as passive agents. Occasionally, two males act alternatively as active and passive agents, though this is rather uncommon.

PSYCHOLOGICAL ASPECTS AND CIRCUMSTANCES OF COMMISSION [9]

i) Penile-anal sexual intercourse in general is viewed by psychologists as a sexual deviation (and not as an offence) that is widely prevalent because it is closest to heterosexuality in offering sexual gratification with a usually willing human object.
ii) Most people have subconscious or unconscious homosexual desires.
iii) Isolation of males from females (or vice versa), as in boarding schools, prisons, defence establishments, ships on long voyages, etc., frequently leads to facultative homosexuality.
iv) History reveals that sodomy enjoyed social acceptability
   - in ancient Greece, where it was quite popular, and from where it spread to Rome, Persia and Arabia
   - in India, China and Japan, where it was alternately accepted and condemned, and
   - in the top echelons of Nazi society.

LEGAL ASPECTS

Although the revelation that they had indulged in sodomy led to the dismissal of several public servants in the United Kingdom, penile-anal intercourse between consenting adults over 21 years of age is now legally permissible in many countries abroad, provided it is practiced in private. Such licence is still not allowed in our country, so that
i) the consent of the passive agent is neither valid nor a defence, and the passive agent will then also be prosecuted;
ii) the marriage contract gives implied consent only for sexual intercourse per vaginum, not per anum; and
iii) under Sec. 13 of the Hindu Marriage Act, conviction for natural or unnatural sexual offences is a valid ground for divorce.

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Homosexual jealousy has often been the motive for suicide or homicide.

CHECKLIST FOR THE MEDICOLEGAL EXAMINATION OF AN ALLEGED VICTIM OF SODOMY/BUGGERY

PRELIMINARY PARTICULARS

1. Written authorisation for the examination from a legally authorized person or agency.
2. General information (name, age, sex, occupation, address of normal residence, marital status).
3. Express written informed consent, duly witnessed.
4. History as per that taken from a victim of alleged rape, modified according to the sex and age of the victim.
5. Gait : Normal, or broadbased and painful.
6. Examination of clothing for
   - damage
   - loose pubic hairs
   - stains of blood/semen/lubricant/faeces.
7. General physical examination, including
   a. at least two marks of identification, and
   b. any injuries indicating resistance.

LOCAL EXAMINATION (in knee-elbow position)

a) **of a victim not used to sodomy/buggery**:
   i) Pain/tenderness during examination.
   ii) Smears of lubricant and loose foreign pubic hairs around/in the anus.
   iii) Perianal abrasions (whose extent and definition are dependent upon the relative proportion between the size of the penis of the assailant and the anal sphincter/canal of the victim, and are therefore most marked in children).
   iv) Occasional perianal bruising, and extension of injuries into the anal canal.
   v) Anal lacerations – Usually triangular and based at the external sphincter, with the sides extending horizontally inward, but rarely more than 2 to 3 cm, because penile penetration would have been limited by a strong contraction of the sphincter ani. These lacerations will result in bleeding onto the thighs and clothing.
   vi) Fresh/dried semen around/in the anus, if present, should be swabbed/scraped off, appropriately packed, sealed, labeled, and sent through the accompanying police constable/investigating officer, to the Forensic Science Laboratory for serological and microscopic examination.
   vii) Digital examination per anum is extremely painful.

b) **of a habitual passive agent :**
   i) Bloodstains are usually not observed.
   ii) Loose foreign pubic hairs and smears of lubricant may or may not be present.
   iii) Perianal hair is usually shaved, and local hygiene scrupulously maintained.
   iv) Perianal skin may be thickened and keratinised from constant friction..
   v) The person does not experience any pain or tenderness during examination, and the external sphincter relaxes reflexly when bimanual traction is applied to the buttocks (*lateral traction test*).
   vi) Funnel-shaped depression of the buttocks towards the anus. (This may not be seen in well-nourished/obese catamites.)
   vii) Anus
      - Dilated and patulous,
      - Loss of rugosity of mucous membrane,
      - Fresh or old fissures/sinuses (tunneling).
   viii) Rectum – Prolapse of mucosa, which is usually thickened, with disappearance of radial folds.
   ix) Evidence of sexually transmitted disease (e.g., condylomata acuminata, chancre/chancroid, gonorrhoeal discharge) may or may not be present.

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x) Digital examination per rectum : Pain is highly improbable when one finger is inserted, and introduction of two fingers may either not be painful, or cause only mild discomfort.

Examination of the alleged assailant/active agent proceeds in exactly the same manner as that of the medicolegal examination of the accused in a case of alleged rape, except that the shaft of the penis may reveal traces of faecal matter and/or lubricant instead of vaginal epithelial cells.

LESBIANISM (SAPPHISM, TRIBADISM)

GENERAL ASPECTS

The counterpart of sodomy as the physical manifestation of male homosexuality, **lesbianism** is the physical expression of female homosexuality. Typically, it involves mutual masturbation, and occasionally an active-passive relationship, by biclitoral/digital/lingual-vaginal stimulation, or the use of vibrators or artificial phalluses (*dildoes*).The terms "lesbianism" and "sapphism" are respectively derived from the island of Lesbos in the Aegean sea (off the coast of Greece), where it was indulged in by an exclusively female population ruled by Queen Sappho. The origin of the word "tribadism" is obscure.

A preferentially active lesbian, who is most often a transvestite or transsexual, is known as a *butch* or *dyke*, while the usually passive agent is called a *femme*.

PARTICULAR LEGAL ASPECTS

In the same way as sodomy/buggery between consenting adults over 21 years of age, lesbianism is licit under the Sexual Offences Act of England. However, under **Sec. 377, IPC**, it is still a criminal offence in India, although publicity and trials are rare, because

- consent is usually mutual,
- such relationships, not being socially acceptable, are carefully concealed, and
- physical or psychological trauma, if any, is relatively minor.

Because habitually active lesbians are strongly averse to normal sexual relationships, (passive agents are usually less so and, in fact, frequently bisexual), continued lesbianism is a valid ground for divorce or annulment of marriage under the Hindu Marriage Act.

Morbid jealousies, as frequent in lesbianism as in male homosexuality, may lead to homicide, suicide, or suicide pacts.

MEDICAL ASPECTS

Lesbianism is extremely difficult, if not impossible to prove, unless

i) Traces of fresh/dried saliva and/or buccal mucosal cells can be detected on/around the external genitalia, or
ii) Injuries result from the use, or forcible introduction into the vagina, of a grossly disproportionate artificial phallus.

BUCCAL COITUS

GENERAL ASPECTS

Also called the "sin of Gomorrah" because of the alleged prevalence of the practice in Gomorrah, the Biblical twin city of Sodom, the term denotes penile/vaginal buccal sexual intercourse, and can therefore be performed by both males and females. Buccal-penile intercourse is called **fellatio**. The partner who performs the act is known as the "fellator"; the one on whom it is performed is the "fellatee." Buccal-vaginal stimulation is referred to as **cunnilingus**.

Although formerly considered as a sexual deviation/perversion, psychiatrists and psychologists now view buccal coitus as a part of normal sexual foreplay.

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PARTICULAR LEGAL ASPECTS

Provided the act is performed by mutual consent of two adults over 21 years of age, buccal coitus is also permitted by the Sexual Offences Act of England.

Under the Hindu Marriage Act, insistence on buccal coitus, if it is non-consensual and repetitive, would constitute a valid ground for divorce.

MEDICAL ASPECTS

The only material evidence of commission of the offence would be

i. spermatozoa of the fellatee in the buccal cavity of the fellator, or
ii. fresh/dried saliva and/or buccal mucosal cells on the penis of the fellatee, or on the external genitalia and/or the vulva of the subject of cunnilingus.

As any of these would be unobtainable under ordinary circumstances (unless the participants were caught in the act), it is generally practically impossible to prove a charge of buccal coitus in a criminal court of law.

BESTIALITY

GENERAL ASPECTS

As the name itself suggests, this unnatural sexual offence involves sexual intercourse with a lower animal.

a) The animals usually selected
   - **by males** are cows, mares, bitches, female sheep/goats/donkeys and large birds; and
   - **by females** (although bestiality in this sex is far less common) are bulls, horses, dogs, and male sheep/goats/donkeys.
b) Just as any animal may be used by a male, so also sexual intercourse may be effected through any of its natural orifices, i.e., the vagina or anus.
c) In our country, bestiality has been depicted prolifically in erotic sculpture, and, in the Kama Sutra, the physical types of men and women taken together with their sexual behaviour have been likened to those of particular animals. Abroad, Greek and Roman mythology, and the literature and rituals of Satanism and Voodoo abound with descriptions of, and references to bestiality.

PSYCHOLOGICAL ASPECTS AND CIRCUMSTANCES

Of all the unnatural sexual offences, bestiality is the least common. It is characteristically the act of an isolated, simpleminded, aberrant farmworker, who is the object of ridicule and/or illtreatment by his fellow men, or by women (in which case he feels frightened and incapable of approaching the opposite sex). He then overcomes his rejection and humiliation by sexual intercourse with an available and usually compliant animal companion. An element of revenge, if present, is usually manifested as sadism (*vide infra*).

Bestiality may also be the product of a superstition among some ignorant males that sexual intercourse with a female donkey cures gonorrhoea !

PARTICULAR LEGAL ASPECTS

Bestiality is a criminal offence worldwide. It is a valid ground for annulment of marriage.

MEDICAL ASPECTS

The offence is difficult to prove (unless the accused is caught in the act), not the least because one of the parties is incapable of expression. The following general findings may be observed on medicolegal examination of the accused :

i) Young adult male, usually mentally challenged.
ii) Injuries inflicted by the animal, if it resisted the act by kicking and/or biting.

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iii) Hair/feathers and/or bloodstains of the animal on the clothing or person of the accused.

SEXUAL PARAPHILIAS
(DEVIATIONS, PERVERSIONS)

These conditions together form one of the groups of psychosexual disorders, invariably featuring involuntarily repetitive, unusual or bizarre imagery or acts, on which sexual arousal and orgasm are dependent. (In simpler terms, *the achievement of sexual gratification by means other than natural sexual intercourse*). What must be remembered is that a single episode, or several with long intervals between them, does not make a person a deviant or a pervert. The terms paraphilia, sexual deviation and sexual perversion connote different perspectives of the same condition. All three are characterized by specialized sexual fantasies (which make up the pathognomonic element), sexual props, and particular requirements of the sexual partner.

However, "paraphilia" emphasizes the unusual quality or nature of the object of an individual's sexual interest; "deviation" refers to non-normative sexual activity in a statistical or cultural sense (i.e., from an epidemiological viewpoint); and the word "perversion" is used when either of the above extends to a shift in the normal psychological-sexual development of the person, i.e., a pathological condition.

Classified Subcategories

1. Fetishism and transvestism
2. Sexual sadism and masochism
3. Exhibitionism and voyeurism
4. Paedophilia
5. Miscellaneous disorders (e.g., necrophagia, necrophilia, etc.)

These various conditions show a tendency to arrange themselves along an object-relations gradient. Thus, fetishism, transvestism, and sexual sadism-masochism have the potential for contact with adult human objects, while paedophilia gives the opportunity for contact with immature human objects. In exhibitionism and voyeurism, there is contact with an adult or immature human object, but this relationship is distanced and strained. Necrophagia and necrophilia involve contact with inanimate (and at times also degraded) human object, and urolagnia, coprophilia, and coprophagia with human excretion and excrement.

FETISHISM (FETICHISM)

a. The essential feature of this condition is the sexual focus on relatively indestructible objects intimately associated with the human body. It is exclusive of transvestism. Sexual activity may be directed toward the fetish itself or may be part of sexual intercourse, and the preferred fetish or class of fetishes is usually constant over a period of time.

b. *Legal aspects* : Fetishism does not ordinarily contravene law, unless
   i) the constant search for a particular fetish leads to theft or mischief, or
   ii) repeated insistence on the use of fetishes by a nonconsenting spouse is cited by him/her as a ground for divorce.

TRANSVESTISM

a. General features of this state (also called "cross-dressing" or **Eonism** after the Chevalier d'Eon, a noted practitioner), which is defined as fantasised or actual dressing in the clothing of the opposite sex, for the purposes of arousal and as an adjunct to masturbation or sexual intercourse, are
   i) a male preponderance, known to commence as early as 16 months of age; and
   ii) commencement of the transvestic syndrome at around puberty (but frank dressing in women's clothing does not begin until mobility

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and relative independence from parents is well established).

Transvestism is also seen in girls, but it seems to start later than in boys, is nowadays easily disguised because of unisex clothing, and has an uncertain outcome.

The diagnosis of transvestism is limited to nonhomosexual, nontrans-sexual men.

b. *Legal aspects*: Transvestism does not cause any problems if it is practiced privately. A male transvestite who flaunts himself in public may be considered to disturb the peace by so doing. In our country, long accustomed to male transvestism under such circumstances as religious festivals and Ramlila performances, and used to the presence of hijras and zenanas, it is unlikely to deserve a second glance. However, if a male spouse persistently indulges in transvestism, even in private, and refuses to participate in normal sexual intercourse, it would obviously constitute a ground for divorce or even annulment of marriage.

SEXUAL SADISM AND MASOCHISM

These represent two opposite and separate aspects of the self , i.e., the active and passive poles of a subjugation-humiliation axis.

The term **sadism**, derived from the Marquis de Sade (who, while in prison, wrote the first of several novels predominantly featuring this practice), is applied to *sexual arousal and orgasm linked to the active infliction, in fantasy or reality, of humiliation, subjugation, or torture of the sexual partner.* The term **masochism**, named after Count Masoch, is the direct reciprocal of sadism, and is defined as *sexual excitement linked with the passive experience of physical or emotional humiliation, subjugation, torture or danger.*

PSYCHOLOGICAL ASPECTS AND CLINICAL FEATURES

i) The power of the sadistic or masochistic fantasy is derived from a poor adjustment to the fear of injury and an angry reaction to it.
ii) Relatively pure sadists and masochists do exist, but, most commonly, participants prefer the active or passive role.
iii) Neither condition is exclusively male or exclusively female, but sadistic practices are more common among men, and masochistic practices are more frequently observed in women.

MEDICOLEGAL ASPECTS

i) Prevalence figures of both sadism and masochism are underestimated, because the acts are usually by mutual consent, and therefore are not reported.
ii) Masochism comes to medical and legal attention when the degree of tolerable suffering is unknowingly exceeded, with tragic results.
iii) A sadist may be sought by law enforcement personnel only in cases of brutality, marital cruelty, rape or *lust murder* (i.e., homicide committed in the pursuit of sexual arousal and orgasm).

EXHIBITIONISM AND VOYEURISM

They are the paired opposites of the **scoptophilic paraphilia**, i.e., sexual gratification in anticipation of the exposure of one's genitalia or observation of the genitalia of another, with orgasm brought about by masturbation during or after the event. A variation of the scoptophilic paraphilia is the "obscene phone call".

Exhibitionism involves repeated acts of exposing one's genitals to a stranger or unsuspecting person. (A male exhibitionist is called a *flasher*).

Conversely, **voyeurism** refers to the repetitive seeking out of situations in which unsuspecting women

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might be observed when they are grooming themselves, undressing, bathing, or copulating. (*Peeping Tom* is the name given to male voyeurs.)

PSYCHOLOGICAL ASPECTS AND CLINICAL FEATURES

Both exhibitionism and voyeurism are almost exclusively male disorders, and are essentially the compulsive repetition of identification with the female sex, and resistance to it by the constant reexamination of one's own genitals and the reaction to seeing them. In both, the preferential objects are well-endowed young women.

LEGAL ASPECTS

Both are criminal offences. Because of repeated exposure, exhibitionists are frequently arrested. Voyeurs are at much less risk, unless they are careless enough to get caught.

PAEDOPHILIA

Paedophilia is defined as preferential sexual activity with children, either in fantasy or reality. It may be performed within the family (amounting to **incest**), or among acquaintance groups, or by strangers. In all cases, the offence is punishable, under **Sec. 376, IPC** if the victim is a female, or **Sec. 377, IPC** if male.

PSYCHOLOGICAL ASPECTS AND CLINICAL FEATURES

i) As with exhibitionists and voyeurs, paedophiles are almost always male. Focussing on children as immature sexual objects, and identification with them compensates for separation and deprivation anxieties that are the root cause of the disorder. Control, domination, and seduction of the child compensate for any sense of powerlessness.
ii) Paedophilia is almost exclusively homosexual (viz., **paederasty**), or heterosexual in nature.
iii) It usually commences as exhibitionism or voyeurism, followed by manipulation by the paedophile of his genitalia (secretly when the child is present, openly once the child has left). Once increasing familiarity and confidence are established, attempts at actual oral, anal, or vaginal coitus begin.
iv) Aggression and sadism are inherent components of paedophilia. These may be subconscious, under control, or open. Only a few paedophilic encounters result in injuries and/or death, but a victim may be badly beaten, or killed, if the assailant is a cold-blooded killer or a lust murderer, or panics after the act.

PARTICULAR LEGAL ASPECTS

i) Among legally identifiable cases, paedophilia is far more common than the other perversions.
ii) Because the victim of the offence is a child, the act is taken far more seriously, and consequently much more effort is taken by law enforcement agencies in arresting and punishing the offender.

NECROPHILIA

Necrophilia is a perversion in which sexual arousal and orgasm can be attained only by sexual intercourse with a corpse, while **necrophagia** is an extreme degree of sadism wherein the sadist derives sexual gratification by tearing out the genitals or other body parts of a corpse and eating them. Both acts are punishable under **Sec. 297, IPC**, (apart from other sections as applicable), which states that *Whoever with the intention of..... (offering) any indignity to any human corpse..... shall be punished with imprisonment of either description for a term which may extend to one year, or with fine, or with both.*

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CHAPTER - 21

Forensic Psychiatry

"To define true madness, what is it, but to be nothing else but mad".

- Shakespeare

DEFINITIONS

Psychiatry deals with the study of mental illness, with particular reference to diagnosis and treatment of mental disorders.

Forensic psychiatry deals with the application of psychiatry in the administration of justice.

Insanity (lunacy, unsoundness of mind, mental derangement)

It is a disorder of the mind or personality in which there is derangement or impairment of mental or emotional components.

SYMPTOMS OF MENTAL ILLNESS

DELUSION

(*Disturbance of thought*)

It is a false belief in something which is not a fact, and which persists even after its falsity has been clearly demonstrated.

Types:

- **Delusion of grandeur** : The patient imagines that he is rich or famous, when he is acutally poor or inconsequential.
- **of Poverty** : Imagines he is poor when in fact he is wealthy.
- **of Persecution** : Imagines that other people are out to harm him.
- **of Reference** : Imagines that people or events relate or refer to him in some speical way.
- **of Influence (control)** : Imagines that his thoughts or actions are controlled by some external agency.
- **of Infidelity** : Imagines that the spouse is unfaithful.
- **of Self-accusation (self reproach)** : Keeps blaming himself for trivial incidents that happened in the past.
- **Nihilistic delusion** : The patient is convinced that nothing exists around him in the real sense of the term.
- **Erotomania (Clerambault-Kandinsky complex)** : Usually seen in women. The patient is convinced that a particular individual (invariably an employer or superior), is in love with her.
- **Pseudologia phantastica** : A variation of this is the *Munchausen syndrome*, in which a person is convinced that he is seriously ill, and goes from doctor to doctor, hospital to hospital, in a vain attempt to diagnose his non-existent illness.
- **Bizarre delusion** : As the term itself suggests, it is an outrageous delusion which can take various forms.

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HALLUCINATION

(*Disturbance of perception*)

It is a false sensory perception without any real, external stimulus.

Types:

- **Auditory** : The patient hears imaginary voices.
- **Visual** : The patient ses non-existent shapes (*unformed images*), or people or animals (*formed images*).
- **Olfactory** : Perceiving non-existent odours.
- **Gustatory** : Perceiving a bad taste in the mouth constantly without apparent cause.
- **Tactile (haptic)** : This may take the form of imaginary sensations over the skin (*formication*), or in a limb that has been amputated (*phantom limb*).
- **Lilliputian (micropsia)** : Perceiving objects to be much smaller than they are.
- **Synesthesia** : A stimulus perceived by a sensory organ other than the one that should actually perceive it, e.g., visualising music, hearing different colours, etc.

ILLUSION

(*Disturbance of perception*)

It is a misinterpretation of a real external stimulus.

OBSESSION

(*Disturbance of thought*)

A single idea or thought is constantly entertained by the patient, which is irrational, but persists in spite of all efforts to drive it from his mind.

IMPULSE CONTROL DISTURBANCE

There is a sudden and irresistible force compelling a person to the conscious performance of an action without motive or forethought.

Types:

- **Kleptomania**: Irresistible desire to steal articles of little or no value.
- **Pyromania**: Irresistible desire to set fire to objects.
- **Mutilomania** : Irresistible desire to hurt or torture animals.
- **Dipsomania** : Inability to stay off alcohol.
- **Trichotillomania** : Irresistible urge to pull out one's own hair.

Box 21.1 explains some common terms used in psychiatry.

CLASSIFICATION OF PSYCHIATRIC DISORDERS

There are two internationally recognised methods of classifying psychiatric disorders – DSM IV and ICD 10.

DSM IV: It is an essentially American system, and is an abbreviation of *Diagnostic and Statistical Manual of Mental Disorders*, IVth edition, 1994. It is a classification prepared and regularly updated by the American Psychiatric Association (APA).

ICD 10: It stands for *International Classification of Diseases, Injuries, and Causes of Death*,* 10th edition, 1992. This classification has been prepared by the WHO, and is periodically updated. Chapter 'F' of the ICD deals with psychiatric disorders.

The ICD-10 classification will be followed in this book, since it has wider acceptance all over the world.

MENTAL AND BEHAVIOUR DISORDERS (ICD-10)

I) **Organic (including symptomatic) Mental Disorders** - The disorders listed in this group have a demonstrable organic cause related to the brain, i.e., there is demonstrable brain pathology.

** Now renamed **International Statistical Classification of Diseases and Related Health Problems.***

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Box 21.1 Some common terms used in psychiatry

Affect : Subjective and immediate experience of emotion at a given time.

Mood : Pervasive emotion or feeling which is sustained.

Cognition : Refers to higher mental functions such as memory, intelligence, concentration, orientation, etc.

Insight : It is to do with awareness of one's own mental condition, and is rated on a 6-point scale, from complete denial of illness to true emotional insight, characterised by significant basic changes in the future behaviour and personality.

Phobia : An irrational fear of an object, situation, or activity.

Panic : An acute, intense, overwhelming episode of anxiety, often associated with feelings of impending doom.

Stupor : A state of akinesis and mutism, with relative preservation of conscious awareness.

Confabulation : A false memory that the patient believes to be true.

Deja vu : A sense of familiarity with unfamiliar surroundings.

Lucid interval : A temporary period of resolution of symptoms in a mentally unsound individual. During this period he will be held responsible for criminal acts.

Neurosis : An emotional disorder in which the patient does not lose touch with reality.

Psychosis : A psychiatric disorder in which the patient loses touch with reality, often experiencing delusions and hallucinations. There is a tendency for violent behaviour.

Psychopath : An essentially normal individual who has deep rooted abnormalities of personality, as a result of which he is unable to conform to conventional standards of behaviour. There is often criminal behaviour, without accompanying feelings of guilt or remorse.

Abreaction : A process of bringing to conscious awareness, previously suppressed unconscious conflicts and emotions. Such a release (*catharsis*) can have therapeutic value.

1. Delirium
2. Dementia
3. Organic amnestic syndrome
4. Other organic mental disorders

II) **Mental and Behavioural Disorders due to Psychoactive Substance Abuse** - This group includes mental disorders arising out of the abuse of psychoactive drugs, e.g., alcohol, cannabis, amphetamines, cocaine, LSD, phencyclidine, etc.

III) **Schizophremia, Schizotypal, and Delusional Disorders** - This group includes psychiatric disorders characterised by disturbances of thought, perception, affect, and behaviour.

1. Schizophrenia
2. Schizotypal disorder
3. Persistent delusional disorder
4. Acute psychotic disorders
5. Schizo-affective disorders

IV) **Mood (Affective) Disorders** - These disorders are characterised by prominent disturbance of mood.

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1. Biploar affective disorder
2. Manic disorder
3. Depressive disorder
4. Persistent mood disorder

V) **Neurotic, Stress-related and Somatoform Disorders** - These disorders were previously loosely termed "neuroses", and are mainly disturbances of emotion.
1. Anxiety
2. Phobic-states
3. Obsessive-compulsive disorder
4. Dissociative (conversion) disorder
5. Somatoform disorders
6. Stress and adjustment disorders.

VI) **Behavioural Syndromes associated with Physiological and Physical Disturbances** - These were previously termed "psychosomatic disorders".
1. Eating disorders
2. Sleep disorders
3. Sexual dysfunctions
4. Puerperal psychiatric conditons

VII) **Disorders of Adult Personality and Behaviour** - These disorders are the persistent expression of an individual's characteristic life style and mode of relating to the others.
1. Personality disorders
2. Impulse control disorders
3. Gender identity disorders
4. Disorders of sexual preference

VIII) **Mental Retardation (MR)** - This group includes disorders with arrested developement of intellect and associated maladaptive behaviour.
1. Mild MR
2. Moderate MR
3. Severe MR
4. Profound MR

IX) **Disorders of Psychological Development** - These disorders have their onset during infancy or childhood and are characterised by an impairment of functions related to biological maturation of the CNS.
1. Speech and language disorders
2. Developmental disorders

X) **Behavioural and Emotional Disorders of Childhood and Adolescence** -
1. Hyperkinetic disorder
2. Conduct disorders
3. Tic disorders

XI) **Unspecified Mental Disorders** A detailed discussion of these psychiatric disorders is beyond the scope of this book. Interested readers are advised to consult standard works on Psychiatry for further elucidation.

CAUSES OF MENTAL ILLNESS

1. **Heredity**	:	Huntington's chorea
2. **Organic**	:	Senility, myxoedema, head injury
3. **Stress**	:	Sudden bereavement, financial loss
4. **Unknown**	:	No cause can be pinpointed for several mental ailments

DIAGNOSIS OF MENTAL ILLNESS

I. **Preliminary Particulars** - Name, age, sex, marital status, education, occupation, income, residential and office addresss, religion, and socio-economic background. In medicolegal cases of psychiatric assessment, it is mandatory to record at least two permanent marks of identification.

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II. **Accompanying Person's Particulars** - The identification particulars of accompanying persons must be recorded, along with details of nature of relationship, whether he stays with the patient or not, etc. When recording the history, it is important to take down the statement of the accompanying person in addition to that of the patient himself.

III. **Presenting Complaints** - Record the details of presenting complaint with particular reference to:

- Onset of present illness
- Duration
- Course
- Precipitating factors
- Aggravating, maintaining, or relieving factors.

IV. **History of Present Illness –**

- When was the last time that the patient appeared to be well ?
- Time of onset
- Record the evolution of symptoms in a chronological manner.
- Record details of disturbances of sleep, appetite, sexual function, etc.
- Enquire about suicidal ideation.

V. **Past Psychiatric and Medical History –**

- History of similar or other illnesses in the past.
- History of serious medical, neurological, or surgical illness.
- History of alcohol or drug abuse.

VI. **Treatment History** - Record details of treatment taken for present and previous episodes, as well as the response.

VII. **Family History** -

Record details of family structure, family history of psychiatric and medical illnesses, family history of drug abuse, attempted or complete suicides, and current social situation of family.

VIII. **Personal History** -

1. *Perinatal history*: Details of pregnancy, date of birth, nature of delivery, perinatal complications etc.
2. *Childhood history*: Who brought the patient up, whether breastfed or bottlefed, relationship to mother, father and other family members, history of neurotic traits (stammering, tics, enuresis, night terrors, head banging), etc.
3. *Educational history*: Age of beginning and completion of formal education, academic achievements, relationship with peers and teachers, truancy, etc.
4. *Play history*: Nature of play activities, friends, relationship with children of opposite sex, etc.
5. *Puberty* : Age at which secondary sexual characteristics developed, details about masturbatory activities, nocturnal emissions (males), menarche (females).
6. *Menstrual and Obstetric history*: Regularity and duration of menses, last menstrual period, number of children delivered, abortions, etc.
7. *Occupational history*: Age at which he started working, jobs held (chronological order), reasons for job changes, job satisfaction, ambitions, relationship with authorities, peers, and subordinates, present income, etc.
8. *Sexual and Marital history*: How was sexual information acquired, masturbatory activities, sexual relationships, nature of sexual activity preferred, gender identity disorder, etc. Also, record details of marriage with reference to date, duration, relationship with spouse, divorces or separations, mode and frequency of sexual intercourse, etc.

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9. *Premorbid personality*: Record details of -
   a. Interpersonal relationships
   b. Use of leisure time
   c. Predominant mood
   d. Attitude to self and others
   e. Attitude to work
   f. Religious beliefs and moral attitudes
   g. Fantasy life
   h. Habits.

IX. **Physical Examination** - Conduct a detailed general physical examination in the usual manner.

X. **Mental Status Examination** -

1. *General appearance and behaviour*:
   a. General appearance: physique, build, height and weight, dress, hygiene, etc.
   b. Attitude: co-operative, evasive, combative, hostile, attentive, interested ?
   c. Comprehension : intact or impaired ?
   d. Gait and Posture : nature of walking, standing, sitting, reclining, etc.
   e. Motor activity : increased/decreased, excited, presence of involuntary movements, restlessness, catatonic signs, etc.
   f. Non-verbal behaviour : nature of eye contact.
   g. Rapport : could or could not be established with the patient ?
   h. Hallucinatory behaviour : talking to himself, odd gesturing, smiling or crying without reason, etc.

2. *Speech*:
   a. Rate and quantity
   b. Volume and tone
   c. Flow and rhythm

3. *Mood and Affect*: *Mood* refers to the pervasive feeling which is sustained, while *affect* refers to immediate and subjective experience of emotion at a given time.

4. *Thought*:
   a. Stream and form: Spontaneity, productivity, poverty of speech, flight of ideas, continuity, tangentiality, etc.
   b. Content:delusions, obsessions, phobias, etc.
   c. Perception: hallucinations, illusions, depersonalisation, etc.

5. *Cognition assessment*:
   a. Consciousness
   b. Orientation
   c. Attention
   d. Concentration
   e. Memory : immediate, recent, remote
   f. Intelligence
   g. Abstract thinking

6. *Insight* : Assess the degree of awareness and understanding of the patient regarding his illness.
7. *Judgement*: His ability to assess a situation correctly and act appropriately.

XI. **Investigations** - Complete medical and toxicological screen, drug levels, electro-physiological tests, brain-imaging tests, neuro-endocrine tests, genetic tests, sexual disorder investigations, etc. Psychological investigations include objective tests, projective tests, neuropsychological tests, rating scales, etc.

XII. **Diagnostic Formulation** - After complete psychiatric assessment, a diagnostic formulation must be made, along with differential diagnosis and treatment plan.

MENTAL HEALTH ACT, 1987

The **Mental Health Act (MHA) 1987**, came into effect only in **April 1993**, in all the states and union territories of India. It replaces the **Indian Lunacy Act of 1912**, which had earlier replaced the **Indian Lunatic Asylums Act of 1858**.

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The MHA is divided into 10 chapters consisting of 98 Sections.

Chapter I deals with various definitions. The Act uses the term 'mentally ill person' instead of the more offensive 'lunatic', and 'mentally ill prisoner' instead of 'criminal lunatic'. Also the term 'psychiatric hospital' is used in place of 'mental hospital.'

A mentally ill person is defined by the Act as *a person who is in need of treatment by reason of any mental disorder other than mental retardation.*

Chapter II of MHA details the procedures for establishment of Mental Health Authorities at Centre and State levels.

Chapter III lays down the guidelines for establishment and maintenance of psychiatric hospitals and nursing homes. There is provision for a Licensing Authority who will process applications for licenses. Private psychiatric hospitals and nursing homes can be run only on a valid license which has to be subsequently renewed every 5 years. An Inspecting Officer will periodically inspect the hospital or nursing home to check for any irregularities.

Chapter IV deals with the procedures of admission and detention in psychiatric hospitals, (**Box 21.2**).

Chapter V deals with the inspection, discharge, leave of absence, and removal of mentally ill persons.

Chapter VI deals with the judicial inquisition regarding alleged mentally ill persons possessing property, and how such property should be managed. If the court feels that a mentally ill person is incapable of looking after himself or his property, an order can be issued for appointing a guardian. If however, it is felt that the person can look after himself, but is only unable to manage his property, a manager can be appointed.

Chapter VII deals with the protection of human rights of mentally ill persons.

Chapter IX outlines the penalties for infringement of guidelines.

Chapter X deals with miscellaneous matters.

MENTAL ILLNESS AND RESPONSIBILITY

The term **responsibility** in the legal sense refers to the liability of a sane person for his actions (or omissions), and consequent punishment if such be contrary to law.

The law presumes every individual to be sane, unless proved otherwise.

CIVIL RESPONSIBILITY

1. **Marriage***: If it is proved that at the time of marriage, one of the parties (spouses) was insane, then such marriage is declared as *null and void*, i.e., as per law the marriage never took place even if consummation had been accomplished. Insanity developing later in the course of married life, which was not present at the time of marriage, may constitute sufficient grounds for *divorce*, but not for *nullity*.
2. **Business contract**: As per the **Indian Contract Act of 1872**, if it is proved that at the time of signing a contract one of the two parties was insane, then the contract becomes legally invalid. However, insanity developing subsequent to a legal agreement will not necessarily invalidate the contract. Also, if at the time of signing, the fact that one of the signatories to the contract was insane was not not known to the other other party, then the contract may not be declared invalid.

** There may be differences among the different Acts in force, e.g., **Hindu Marriage Act, Dissolution of Muslim Marriages Act, Parsi Marriage and Divorce Act, etc.***

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Box 21.2 Procedures of Admission and Detention in Psychiatric Hospitals (**Restraint of the Insane**)

Chapter IV of the **Mental Health Act, 1987**, lays down guidelines for psychiatric hosptials in relation to the admission, detention, and discharge of mentally ill persons.

Admission -

I. *Voluntary admission:*
   1. On patient's request, if he is a major.
   2. On guardian's request, if he is a minor.

II. *Admission under special circumstances:*
   This is a type of involuntary hospitalisation when a mentally ill person cannot express his willingness for admission. A relative or friend of his can apply in writing for admission, and the medical officer in charge of the hospital can do so, if he is satisfied that the admission would be in the best interests of the patient. The duration of admission cannot exceed 90 days.

III. *Reception order on application.*

IV. *Reception order without application :*
   This is done in the case of wandering, dangerous, neglected, or illtreated mentally unsound individuals. The patient must be produced before a Magistrate.

V. *Admission as inpatient after judicial inquisition.*

VI. *Admission as a mentally ill prisoner.*

Detention -

A magistrate can order the detention of an alleged mentally ill person for short periods pending report from the medical officer concerned, or pending his removal to a psychiatric hospital or nursing home. In the former case, the period of such detention cannot exceed 10 days, while in the latter case, maximum period permissible is 30 days.

Discharge -

1. A voluntary patient must be discharged within 24 hours of receipt of request for discharge made by the patient himself, or his guardian.
2. A mentally ill person admitted on application can be discharged on his own request, or the request of a friend or relative.
   In these cases, it must first be made certain that the patient has been cured of his illness, and that he is fit to be discharged.
3. The officer in charge of a psychiatric hospital can order the discharge of any inpatient, on the recommendation of two medical practitioners one of whom should be a psychiatrist.
4. A relative or friend of a mentally ill person can make an application for his discharge even if he is not fully cured, provided he is not dangerous to society. In such a case, the patient can be discharged on the guarantee of the relative or friend that he would take care of the patient. Execution of a bond attesting to this is necessary.
5. A person detained on a reception order can be discharged, if a judicial inquisition finds him of sound mind.

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For the purposes of a contract, a person is said to be of sound mind if at the time of making the contract, he is capable of understanding it and of forming a rational judgement.

3. **Management of property** : If a person who owns property becomes insane and is incapable of managing his affairs with sound judgement, any relative or friend can approach the court for a judicial inquisition. If it is proved that the person is insane in the course of such inquisition, then the court can appoint a Guardian or Manager depending on the circumstances (See page no. 250).

4. **Capacity to depose as witness**: As per **section 18** of the **Indian Evidence Act**, an insane person is not competent to give evidence if he is incapable of understanding the questions put to him, or giving rational answers to them. However, such a person can give evidence during a lucid interval, depending on the discretion of the judge.

5. **Testamentary capacity**: Detailed in **Box 21.3**.

Box 21.3 Testamentary Capacity

Testamentary capacity refers to the capacity of a person to make a valid will.

A **will** is a document detailing the disposition of property owned by a person, which is prepared by him during his lifetime but takes effect only after his demise. The person who makes the will is referred to as testator. It can be revoked or changed any number of times (at his own sweet will!).

Eligibility for making a will: As per the **Indian Succession Act, Section 59**, the following persons are eligible to make a valid will -

- Every person of sound mind who is over the age of 18 years.
- An insane person cannot write a valid will, unless he is in a lucid interval.
- An intoxicated person cannot make a will, unless it is certified by a doctor that he was in possession of his senses.
- A deaf, dumb, or blind person can make a will if he can communicate effectively.
- Convicts are not debarred from making a will.

Conditions: The person making a will should

- Have a sound disposing mind.
- Have thorough knowledge about his wealth and property.
- Be free from undue influence, coercion, or fraud.
- Do it voluntarily.

Procedure: The will must always be in writing. The only exception provided for under law, is for members of the armed forces who are out on an expedition or engaged in warfare. They can make an oral will (*privileged will*). Also, muslims are permitted to make an oral will by their personal law.

There is no particular format for a will as per law. In fact, it need not even be executed on a stamp paper. Typing is desirable, but not essential. The testator can write a will himself, using a fountain pen or ball pen (*holograph will*).

The will must be attested by at least two witnesses, neither of whom can be beneficiaries. It is preferable that one of them should be a doctor. The signature or thumb impression of the testator himself is ofcourse mandatory.

The will comes into effect only on the death of the testator.

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CRIMINAL RESPONSIBLILITY

The entire concept of criminal responsibility revolves around the principle of *mens rea* or *guilty mind*. This is itself a component of a legal dictum (in Latin) : *Actus non facit reum, nisi mens sit rea.* When translated, it reads: "The physical act alone does not make a person guilty ; the mental component in the form of evil intent (guilty mind) is equally important."

Since an insane person cannot comprehend the nature of his own actions, or that they may be contrary to law, he is exempted from criminal responsibility. However, insanity per se is not enough to render a person innocent (of his alleged criminal action). He must satisfy certain conditions which are laid out in law. In India, **section 84** of the **Indian Penal Code** outlines the guidelines applicable to criminal responsibility of insane individuals. This is an offshoot of the **McNaughten Rule** which came into force in the UK in the 19th century. Daniel McNaughten was a psychotic suffering from delusions of persecution, who was convinced that his problems would end only with the demise of the then Prime Minister of Britain, Sir Robert Peel. In January 1843, he lay in wait for the Prime Minister, and when he encountered him, fired at him with his revolver. But because of mistaken identity, the secretary to the Prime Minister, Edward Drummond was assassinated, and Sir Robert Peel escaped. McNaughten was arrested and tried, but the jury after testification by 9 physicans, found him "not guilty by reason of insanity". The people were outraged, as also Queen Victoria and Sir Robert Peel himself. As a result, 15 prominent judges were invited by the House of Lords. They were asked to respond to a series of questions on criminal responsibility. The answers were subsequently condensed and put forth as the McNaughten Rule.

Section 84, IPC:

Nothing is an offence which is done by a person, who, at the time of doing it, by reason of unsoundness of mind, is incapable of knowing the nature of the act, or that he is doing what is either wrong or contrary to law.

In order to apply S.84 in a given case,it has to be clearly established that at the time of committing the offence, the accused was labouring under a defect of reason which had been caused by unsoundness of mind, with the result that he was rendered incapable of knowing the nature of the act, or that he was doing what was wrong or contrary to law.

A plea of insanity at the time of the trial will not help the accused. Two things have to be, therefore, clearly established, i.e., that at the time of committing the crime the accused was of unsound mind, and that the unsoundness of mind was of a degree and nature which rendered the accused incapable of knowing the nature of the act, or that he was doing what was either wrong or contrary to law.

It is important to note that "legal insanity" is not the same as "medical insanity". While the latter follows the classification laid down in psychiatry, "legal insanity" is mainly with reference to mental deficiency and psychotic conditions. It does not recognise most of the neuroses and personality disorders as evidence of insanity.

It is also important to remember that it is the state of mind of the accused at the time of the commission of the offence that matters, and not at any other time before or after the event.

The burden of proving insanity is cast upon the accused, and he must prove that at the time of the incident he was by reason of unsoundness of mind incapable of judging the nature of his act (**S.105,**

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Indian Evidence Act). Every man is presumed to be sane and to possess sufficient degree of reason to be responsible for his crime unless the contrary is proved.

One major defect in the McNaughten Rule (which in turn is reflected in S.84 IPC), is that emotional aspects relating to unsoundness of mind are not given due weightage. Hence some of the major psychiatric disorders, especially neurotic disorders, are not given any importance. This aspect has been attempted to be rectified in Western countries such as the UK and the USA, by way of new guidelines:

- **Durham Rule (1954)**
- **Curren's Rule (1961)**
- **American Law Institute's Test**

Role of Medical Officer

In a criminal case where insanity is pleaded as a defence, the opinion of a psychiatrist is necessary to prove its existence in the accused. If he is requested by the authorities to examine the accused and furnish his opinion, he must proceed cautiously and meticulously. Some helpful pointers to an insane crime include the following :

- History of mental disease in the family of the accused.
- Lack of motive in the commission of the offence.
- Lack of premeditation/pre-planning.
- Absence of accomplices.
- No attempt to destroy evidence, or to flee from the scene of crime.
- Ghastly or bizarre nature of crime.

Box 21.4 presents a classical case of a psychotic killer.

FEIGNED INSANITY

Since the law provides for exempting a criminal from punishment if he was insane at the time of the

Table 21.1 Differences Between True and Feigned Insanity

Feature	True insanity	Feigned insanity
Onset	Gradual and insidious	Abrupt and dramatic
Motive	Absent	Present (commission of offence)
Precipitating factors	May be present (stress, financial losses, bereavement, etc)	Absent
Symptomatology	Conforms to a particular type of psychiatric disorder	Usually does not conform to any known type of disorder, and is often exaggerated Symptoms may be absent when the patient is not under observation
Facial expression	Usually listless, vacant, and fixed	Frequent changes, exaggerated grimaces.
Insomnia	Often present	Cannot withstand lack of sleep for more than a night or two
Exertion	Can withstand fatigue and hunger for prolonged periods	Cannot withstand fatigue or hunger for more than a day or two
Personal hygiene	Does not pay attention to his personal hygiene	May only pretend to be disinterested in appearance or dress
Frequent psychiatric examinations	Does not mind	Resents, since he fears detection

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Box 21.4 The Vampire Killer

The first murder occurred on 20 December,1977. A 51 year old man who was retrieving groceries from his car outside his home in Sacramento, California, was shot twice in the chest by an unknown assailant. The murder was without any motive. The assailant was Richard Trenton Chase, a young psychotic male. But the police were clue-less.

On January 23, 1978, Chase burglarised a home, stealing 16 dollars in cash, a pair of binoculars, and a dagger. He also urinated into an open drawer of undergarments and defecated on a bed. He then proceeded to a supermarket where he came across a woman he had known in high school. She was horrified by his appearance - unkempt, straggly hair, dirty hands, and yellow crust around his lips. She figured he was either crazy or on drugs, and made a quick getaway.

Later the same day, Chase shot dead a 22 year old woman as she came out of her home carrying some garbage bags. He then savagely mutilated her body, and dipping his hands in her blood, smeared his face with it, and licked his fingers. Not satisfied, he fetched a paper cup from her kitchen, scooped blood into it and drank it.

The Sacramento police department was completely baffled by this vicious homicide. The neighbourhood of the crime was thoroughly canvassed for information. Investigators learned that there had been a burglary on the same block earlier that day, which appeared to be related.

On January 27, 1978, the police were called to a residence in the same general area, on a report of a multiple murder. A neighbour discovered the whole family murdered when she went on a casual visit. The woman of the house who was 36 years old, had been shot three times and her body was eviscerated. Her 52 year old male companion had been shot in the head, as also the woman's 6 year old son. A 22 month old baby, who the woman was baby sitting was missing from a bloodstained crib. There was also evidence of sodomy (of the woman) and anthropophagy, since some body parts and organs were missing, apparently cut out with a knife. A search of the rest of the house revealed bloodstained water in a tub in the bathroom, and pieces of human entrails.

By now it was clear that these series of murders had been committed by the same assailant who was obviously quite disturbed. Forensic psychologists profiled the suspect to be a young white male in his twenties, probably schizophrenic, a loner type of individual, unmarried, unemployed or working as a menial labourer, and living within a 1-mile radius of the crimes. An extensive canvas operation was initiated. On January 28, a witness reported seeing Richard Chase a few days earlier in a supermarket, from whom she had in fact fled. She told police that he appeared crazy enough to be considered as a suspect. Investigators were despatched to Chase's apartment. There was no response when they knocked on his door, though they could hear somebody moving inside. Suddenly the suspect burst through and began running away. The police officers chased him and wrestled him to the ground. They were horrified to discover that he was carrying fast food containers stuffed with human body parts and blood. He was also carrying a gun in a shoulder holster. Subsequent search of his apartment revealed extensive evidence of all the murders. There was also ample evidence that he had drunk his victims' blood, and cooked and eaten their body parts. The refrigerator was stocked with human organs.

Psychiatric examination of Richard Chase disclosed the reason for his gruesome activities. He was convinced that alien flying saucers were drying up his blood through some sort of radiation, and therefore in order to survive he had to constantly replenish his supply. Chase was sent for trial, but while being incarcerated in his cell, he overdosed on psychiatric medication and killed himself.

Courtesy of Lieutenant Ray Blondl, Commanding Officer, Homicide Bureau, Sacramento County, California, USA.

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offence (though he may be incarcerated in a psychiatric hospital for treatment), it is not uncommon for an offender to plead insanity as a defence specially in cases of murder. Such a defence may be put forth even though the culprit is actually sane, so that he may escape stiff punishment such as life imprisonment or death. In order to substantiate his claim, the person may pretend to be insane by manifesting fabricated signs and symptoms. This is refered to as **feigned insanity**. The court or the police may then request for the opinion of a psychiatrist to clarify the issue. When called upon to do so, the psychiatrist must meticulously examine the individual and pay special attention to certain clues which could indicate pretence ("feigning or shamming"). Some of these are mentioned in **Table 21.1**.

The question of criminal responsibility may arise in a number of situations and conditions which are ambiguous, posing difficulties for the medical officer as well as the investigating authorities. **Box 21.5** outlines the current status with reference to these conditons.

Box 21.5 Criminal responsibility In some special situations

1. **Automatism**: This refers to a condition in which the conscious performance of an action is impaired to such an extent that the patient does not remember anything subsequently. This may occur after an epileptic fit, or an episode of concussion, or even hypoglycaemia.

   The person is usually not held responsible for his actions.

2. **Somnambulism**: This refers to the condition in which a person walks about in his sleep, and is therefore also called "sleep walking". Such individuals may not really be abnormal, though some do suffer from psychiatric problems. There is often some stressful episode immediately preceding the walk.

   The person is usually not held responsible for any action committed during the episode of sleep-walking.

3. **Somnolentia (Semi-Somnolence)**: This is a condition midway between sleep and wakefulness, as for instance when a deeply slumbering person is rudely awakened. In the brief period during which he is confused, he may strike out reflexly at somebody who is near him, or who is in fact shaking him awake.

   The person may not be held responsible for his actions during this "twilight period".

4. **Hypnotism or Mesmerism**: This is a form of sleep-like trance induced in a person by a process of suggestion. A hypnotised person may be subsequently induced into the performance of actions which he does not remember afterwards.

   Hypnotism and mesmerism cannot be pleaded as defence in the commission of crimes. Both the hypnotiser and the hypnotised may be held liable.

5. **Drunkenness**: If a person voluntarily consumes an intoxicating drink and commits a crime under its influence, he is held responsible for his action (S. 86 IPC). But if the drink was administered without his knowledge or against his will, he will not be held criminally responsible (S. 85 IPC).

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CHAPTER - 22

Trace Evidence: Biological Materials

"Whenever two objects come into contact with each other, there is a mutual transfer of material from one object to the other".

- Sir Edmund Locard.

The above famous quote from an eminent expert underlines the main principle of Forensic Science, which constitutes the basis of all crime detection. Since awareness among criminals has increased regarding scientific investigation of crime, the forensic scientist is left only with microtraces of evidence in many modern day offences. That is why in recent times, several technologically advanced instruments and methods have been developed, to detect such microtraces of evidence.

Biological materials such as blood and other body fluids – semen, saliva, sweat, etc., are well known examples of trace evidence which are commonly encountered in scenes of violent crime. A clever criminal may try to clear the scene of crime in an attempt to obliterate such evidence, so much so that only microtraces may be left behind.

The routine use of ultraviolet light can help in detecting stains of various origin such as blood, semen, saliva, pus, milk etc., as they all fluoresce under light.[1]

IDENTIFICATION OF BLOOD AND ITS STAINS[2]

Testing for the presence of blood is perhaps the most important step in the examination of a stain associated with a violent crime. Unless this issue is resolved, it may be futile to proceed with further analysis.

Identification of blood is based upon the presence of blood cells or compounds characteristic of blood, such as

a) erythrocytes and leukocytes
b) blood serum proteins
c) haemoglobin and its derivatives.

Microscopy

The observation of a fresh blood stain under the microscope would reveal the presence of erythrocytes and leukocytes. Apart from identification of blood, microscopic examination may also reveal other information such as the species origin, sex of the individual (on the basis of Davidson body, *page no. 38*), and evidence of disease, e.g., sickle shaped erythrocytes indicate sickle cell anaemia.

Chemical Tests

In case blood stains are rendered invisible by washing of the crime scene (in an attempt to obliterate the stains), the suspected area can be sprayed with **luminol**, which would make the entire area glow in the dark, if blood is present. Luminol, i.e., 3-amino pthalylhydrazide, is a colourless substance which reacts with blood in the presence of an oxidant to form a product, which luminesces in the dark.

In the case of a visible stain, it can be subjected to various chemical tests such as **benzidine test, phe-**

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Table 22.1 Colour Tests (Catalytic Tests) for blood

S.No.	Reagent	Colour	Sensitivity
1.	Benzidine in glacial acetic acid	Blue	1:3,00,000
2.	Phenolphthalein (reduced with 20% KOH) in ethanol	Pink/Purple	1:5,000,000
3.	O-toluidine	Blue	1:300,000
4.	Leucomalachite green with sodium perborate and glacial acetic acid	Green	-

nolphthalein (Kastle-Meyer) test, o-toluidine test, leucomalachite green test, etc. All these tests make use of the peroxidase-like activity of the haeme group of haemoglobin, which in the presence of an oxidant like hydrogen peroxide oxidises a colourless material such as phenolphthalein, benzidine , o-toluidine etc., to a coloured one. The colours produced by various reagents are mentioned in **Table 22.1**.

Benzidine is widely used in spite of its carcinogenicity, since the reagent is easy to prepare, and the test is easy to perform and is highly sensitive.

Though these reagents are highly sensitive to minute traces of haemoglobin and its derivatives, they are not specific to blood, since certain chemical oxidants and catalysts (such as copper and nickel salts, rust, paint, etc.) , peroxidase present in plant sources (such as apple, potato, onion, tomato, cabbage, etc.) and other body fluids (such as saliva, CSF, pus, etc.) also give false positive reaction, especially with benzidine. Hence these tests are considered purely preliminary in nature, and the presence of blood in the suspected stain has to be confirmed by other methods.

In general, a negative result provided by the Benzidine test is said to indicate the absence of blood in the stain. But, recently, in a preliminary communication by **Pascual & Grifo**, a hypothesis has been put forward that in the presence of a substance with a high reduction power, the oxidising potential of blood may not become apparent, which can lead to a false negative result with the Benzidine test. In order to validate their hypothesis, they mixed lemon juice, which has a high reduction potential, with blood in various concentrations, and demonstrated negative results.[3] This casts serious doubts on the reliability of Benzidine test (as well as the other catalytic tests), as a screening method for blood.

Confirmatory Tests

1. **Crystal Tests (Fig 22.1)**: All the crystal tests are based upon the formation of haemoglobin-derived crystals such as haematin, haemin, haemochromogen, etc.

   - **Teichmann Test (Haemin crystal test)**
     When blood is heated with glacial acetic acid in the presence of a salt like Nacl, dark brown rhombic crystals are formed due to the combination of halogen with ferriprotoporphyrin. Overheating, presence of rust, or any other substance which degrades haemoglobin or haematin to haematoporphyrin will interfere with crystal formation.

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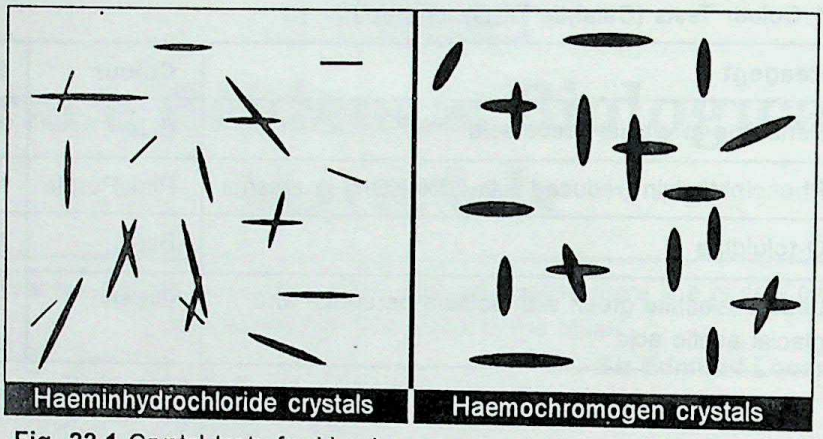

Fig. 22.1 Crystal tests for blood

- **Takayama Test (Haemochromogen crystal test)**
  When blood is heated with Takayama reagent (pyridine-5 ml, NaOH–5 ml, glucose–5 ml, and distilled water-16 ml), pink, rhomboidal, haemochromogen crystals are formed.

  Both these tests are highly specific for blood, and require as little as 0.001 ml of blood or 0.1 mg of haemoglobin.

2. **Spectrophotometry** (Fig 22.2): Haemoglobin and its derivatives exhibit characteristic bands of absorption at specific wavelengths which can be observed by a spectrophotometer. Haemoglobin and its derivatives show a common absorption band at 400-425 nm (*Soret band*), apart from one or more bands at their characteristic wavelengths. Hence the presence of two or more absorption bands in the spectroscope confirms the presence of blood in the stain.

   Porphyrin compounds and their derivatives from other animal and vegetable sources may share a common band with haemoglobin and its derivatives. Hence, a single absorption spectrum should never be relied upon for confirming the presence of blood.

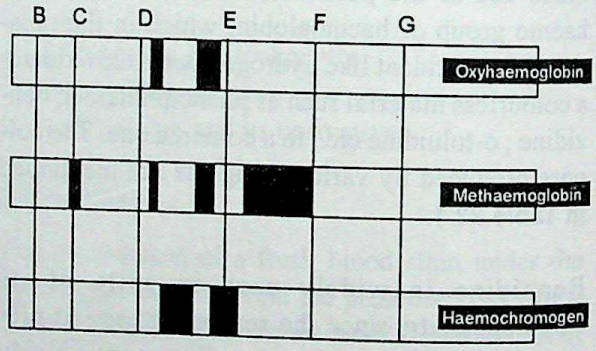

Fig. 22.2 Spectroscopy of blood: Absorption bands

3. **Electrophoresis**: Haemoglobin and its derivatives are made up of conjugated proteins and carry a specific charge, which at a particular pH, can be separated by induction of an electric field on a suitable medium like agarose gel. The distance travelled by each is characteristic for that particular substance, and can be detected on the gel medium by spraying any one of the common colour reagents (benzidine, leucomalachite green, etc). The false positives given by the colour tests are obviated in this procedure, since such substances either carry no charge, or possess a different charge other than haemoglobin and its derivatives, and hence would not be separated by electrophoresis. This

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makes the method a reliable confirmatory test for blood.

Immunoelectrophoresis further enhances the reliability of the above method by combining immmunodiffusion with electrophoresis. In this procedure, the extract of the stain is subjected to electrophoresis and then the slide is placed in a well which contains antiserum. The antiserum diffuses into the gel, and there is migration of serum proteins towards the diffusing antiserum forming a white line of precipitation of Ag-Ab complex, whereas haemoglobin and its derivatives remain at the point of origin giving a pinkish ring around the sample well. The white precipitin line of Ag-Ab reaction, and the pink ring of haemoglobin or its derivatives, constitute double confirmation for the presence of blood.

4. **Chromatography**: The principle of this method is the separation and migration of haemoglobin and its derivatives in a suitable stationary phase such as silica gel, alumina, etc., (or paper, in paper chromatography), with respect to a particular solvent system. The distance travelled by Hb and derivatives in relation to that particular solvent system (termed as the R_f value) is a highly characteristic feature aiding in their identification. The separated derivatives can be detected on the TLC (thin layer chromatography) plate by either uv radiation or by the use of appropriate colour reagent.

Determination of Species Origin

After identifying the stain as blood, the next step is to find out the species, as to whether it is human or animal. Many immunological tests are used to determine this.

When the antigen derived from a particular species is injected into the body of another, the latter recognises it as a foreign body, and produces antibody (antiserum). This antiserum forms an antigen-antibody (Ag-Ab) complex with the antigen, both *in vivo* and *in vitro*. The white precipitin formed by the Ag-Ab complex *in vitro*, forms the basis for the following tests employed in the determination of species:

1. Ring test
2. Gel diffusion
3. Immuno electrophoresis
4. Crossed-over electrophoresis
5. Latex particle method

Antihuman sera prepared from horses (H type), and rabbits (R type), are generally used to determine the blood of human origin.

Grouping of Blood Stains

The common blood groups such as ABO, MNS and Rh systems are determined in a blood stain by one of the following methods.

1. Latte's crust method
2. Absorption - elution technique
3. Absorption - inhibition
4. Latex method, etc.

Whereas absorption–elution technique is suitable for small stains, the absorption –inhibiton technique is less susceptible to interferences. An aberrant reaction relating to the presence of B factor neither belonging to the victim nor the assailant, has been observed in the absorption–elution method by some investigators.[4]

Individualization of blood stains: The various **blood group systems** (ABO, Rh, MNS, Kell, Duffy, Lutheran, Kidd, etc.), polymorphisms exhibited by **serum proteins** (Hp, Gc, Ag, Gm^1, Gm^2, KM etc.), and **red cell enzymes** (PGM, EAP, GPT, EsD, ADA, AK, GLO-I, etc) can help in individualising blood stains to great extent. **HLA typing** and **DNA fingerprinting** can accomplish this beyond any doubt.

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Table 22.2 Inheritance of Blood Groups

Phenotypes of Parents	Phenotypes of Children	
	Possible	*Impossible*
O x O	O	A,B, AB
O x A	O, A	B, AB
O x B	O, B	A, AB
O x AB	A, B	O, AB
A x A	O, A	B, AB
A x B	O, A, B, AB	None
A x AB	A, B, AB	O
B x B	O, B	A, AB
B x AB	A, B, AB	O
AB x AB	A, B, AB	O
M x M	M	N, MN
M x MN	M, MN	N
M x N	MN	M, N
MN x MN	M, N, MN	None
MN x N	N, MN	M
N x N	N	M, MN

Disputed maternity and paternity: The blood group systems and the polymorphisms of serum proteins, red cell enzymes, and HLA and DNA typing mentioned above, are all inherited from the parents and follows Mendelian laws of inheritance. This fact enables the forensic scientist to determine the parentage of a child in disputed cases. **Table 22.2** mentions salient principles with reference to inheritance of blood groups.

Apart from being an identification marker, a blood stain can also give information regarding the following:

1. **Source of blood**: Foetal blood can be distinguished from adult blood by electrophoretic determination of HbF or L-foetoprotein (AFP).

Blood of pregnancy and abortion/parturition can be differentiated by the presence of chorionic gonadotropin (in the earlier stages), or heat-stable alkaline phosphatase (in the later stages). Detection of certain types of alkaline phosphatase and placental lactogen (from 15th week of pregnancy), would enable the identification of placental blood.[4]

Menstrual blood is detected on the basis of absence of fibrin content in it.

Arterial and venous blood can be differentiated to some extent on the basis of amount of blood loss and spurting characteristics. Arterial blood loss is generally copious in nature, bright red in colour, and shows spurting due to high pressure

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and velocity, whereas venous blood is dark in colour, and oozes out gradually, as a result of which there is no evidence of spurting.

Blood which has effused during life can be peeled off in scales upon drying, due to the presence of fibrin, whereas blood which has flowed after death tends to break up into a powder on drying.[5]

2. **Direction of fall of blood**: A trail of blood stains, or splashes of blood on walls or furniture may be encountered in a scene of crime. It is important to determine the height and direction of fall of blood in such cases. If blood falls vertically from a realtively low height (not exceeding a few centimetres), the drops are round and sharply delineated. If height is increased above 30 cm, the spots show prickly edges, the projections growing finer and larger in number with increase in height. Splashes of blood striking a surface obliquely may appear like spears or exclamation marks, depending upon the velocity and angle of fall ; the pointed end indicates the direction of motion (**Fig. 22.3**). This is especially important in assessing the relative positions of victim and assailant.

3. **Age of stains**:

Several points can help in dating blood stains:

1. Progressive decrease in solubility of blood stain in various aqueous solutions, with age.
2. Change in colour from red to brown due to oxidation of haemoglobin to methaemoglobin.
3. Measuring colour changes by colourimetry and spectrophotometry.
4. Immunoelectrophoresis.
5. Changes in enzyme activities of blood cells.
6. HPLC - in old stains, peaks corresponding to haeme, and a and b chains, have been detected at 220 nm with a retention time of 7 minutes, which are not seen in fresh blood, and the height of peak increases with age.[6]

4. **Sex of the individual**:

The presence of Davidson body or Y chromosome in WBCs, can help in sexing blood stains.

IDENTIFICATION OF SEMINAL STAINS

Identification of analytes characteristic of semen is particularly useful in the investigation of sex crimes. Semen is a suspension of spermatozoa in seminal plasma which is a pool of different secretions by different structures and glands such as vas deferens, seminal vesicles, prostate gland, and mucous glands. Seminal plasma consists of a very high concentration of citric acid, fructose, zinc, phosphoryl choline, glyceryl phosphoryl choline, spermine and spermidine, acid phosphatase, y-seminal protein, 19-OH prostaglandin, etc. Identification of seminal stain is based on the detection of any of these constituents.

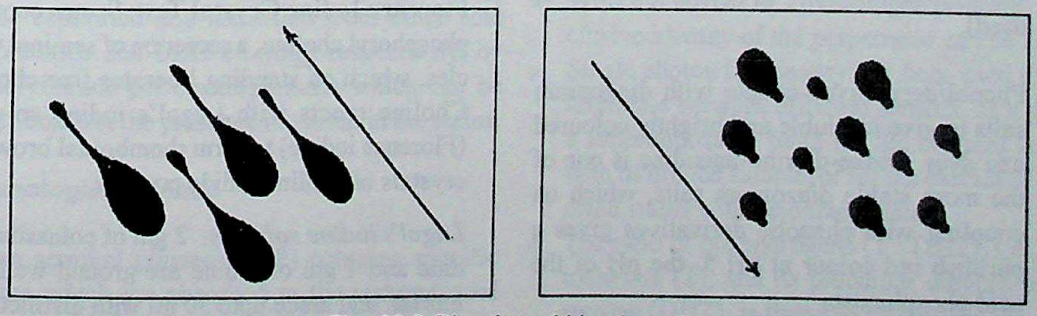

Fig. 22.3 Direction of blood spatter

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Invisible stains can be detected by examining the material in question under uv light. Seminal stains fluoresce bluish-white in colour on a dark background. This is true of other body fluids also such as blood, saliva, pus, milk, etc. Since certain fabric whiteners also fluoresce under uv light, it might not be reliable if the stain is present on cloth, and has to be subjected to other tests characteristic of semen.

Chemical Tests

The seminal stain is extracted by soaking a small bit of the stained material in sufficient quantity of sodium acetate buffer (pH 4.5). The extract is subjected to various tests :

1. **Acid phosphatase Test**: Acid phosphatase is a prostatic gland secretion found in very high concentration in semen when compared with other body fluids. Semen recovered from vagina retains recognisable amounts of acid phosphatase upto 36 hours. Dried stains which are not decomposed, retain acid phosphatase acitvity for weeks or months, whereas heating the specimen above 60° C destroys the activity.

   Acid phosphatase, on addition of p-nitrophenyl phosphate, releases paranitrophenol which can be detected by the following two methods[7]:
   i. Phenol and its derivatives give a yellow colouration in the presence of an alkaline solution, (sodium carbonate is commonly used).

   ii. Phenol/derivatives couple with diazonium salts to give insoluble and brightly coloured azo dyes. Diazo-diortho-anisidine is one of the more stable diazonium salts, which on coupling with phenolic derivatives gives a purplish red colour at pH 5, the pH of the acid phosphatase.

   Many other organisms and materials such as bacteria, snake venom, almonds, cauliflower, etc., contain acid phosphatase which can interfere with the screening of seminal fluid or stain.

   Cauliflower juice and the extract of *Ulex europeus* (gorse seeds), give reactions as intense as that of human semen.[7] Hence a qualitative acid test is insufficient to prove semen in the absence of other independent tests. Various analytical methods have been devised to measure the level of acid phosphatase activity of endogenous and post-coital vaginal washings.

2. **Zinc Test**: The concentration of zinc in semen is unusually high (140 mg/ml in contrast to 1.2 mg/ml for normal blood serum).[8] Zinc, a secretion of prostatic gland is supposed to maintain microbiological sterility. Reagents which give brightly coloured products in the presence of zinc are used to distinguish semen from other body fluids. Pyridylazonaphthol (PAN) reagent was used by **Suzuki et al.**, who suggested for the first time that the high concentration of zinc could be used as a marker for semen.[9] Laboratory-based methods use atomic absorption spectrometer for quantitative detection of zinc in semen.

   Acid phosphatase degrades slowly in moist samples, whereas zinc does not, making it a more useful parameter in the case of older specimens.

3. **Florence Iodine Crystal Test**: Semen contains phosphoryl choline, a secretion of seminal vesicles, which on standing liberates free choline. Choline reacts with Lugol's iodine solution (Florence iodine) to form rhomboidal brownish crystals of choline iodide crystals.

   Lugol's iodine solution: 2 gm of potassium iodide and 1 gm of iodine are ground well in a mortar, and made upto 30 ml with distilled water.

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4. **Barberio's Test**: Semen contains spermine, a prostatic gland secretion, which reacts with picric acid to form needle-shaped yellowish crystals of spermine picrate.

Microscopy

The presence of semen in a stain can be considered a certainty only if spermatozoa or sperm heads are detected microscopically. A smear of the seminal extract on a microscope slide is stained with haemotoxylin or Ziehl-Neelsen's stain, and viewed through an oil immersion microscope.

Microscopic visualisation of spermatozoa is still considered to be confirmatory of semen, but can show false-negative results for a variety of reasons such as azoospermia, vasectomy, or old age. In such cases, the stain can be reported to be semen if it gives positive results with Florence test, human precipitin test, and acid-phosphatase test.[7]

According to a more recent finding, a negative test obtained from the parallel combination of the zinc test (with modified PAN reagent) and the acid phosphatase test, predicts the absence of spermatozoa by microscopical examination very well, and hence can help in sorting out materials which deserve further investigation by more elaborate techniques.[10]

Electrophoresis

Acid phosphatase and LDH-x isoenzyme of semen can be separated and identified from other acid phosphatases and LDH enzymes respectively, by polyacrylamide gel electrophoresis which can be used to confirm the presence of semen in the stains.

Immunological Method

Human seminal plasma (HSP) contains specific antigens which are absorbed to the spermatozoal surface and are called as sperm-coating antigens. They can be detected both on the sperm cells and in the ejaculated fluid.

The following antigens have been identified : the specific antigen P-30 (also called *prostate specific antigen*) produced by the epithelial cells of the prostatic gland,[11] MHS-5, originating from the epithelial cells of seminal vesicles,[12] and an antigen corresponding to Mab 4E6.[13] Antibodies specific to these antigens can be detected by various immunological tests such as immunodiffusion, ELISA, agglutination–inhibition of HSP-coated latex microspheres, dot-immunobinding test, etc.

These HSP antigens have been reported to be present in the semen of aspermic persons as well, making them a very reliable confirmatory test for semen.

Determination of Species

Semen originating from various animal species as well as humans, can be differentiated by a number of immunological methods based on the precipitin reaction, (same as for blood, *vide supra*). LDH isoenzyme pattern, presence of Y-body in the sperm, and human seminal plasma- specific antigens, are highly characteristic of human semen.

Individualization of seminal stain

- ABO grouping, PGM, AK, HLA, LDH, Gm & Km, GLO, diaphorase patterns, etc., can all be demonstrated in the semen of secretors.
- With the advent of DNA-finger printing, conclusive identity of the perpetrator can be fixed.
- Single photon fluorimetry has been used to differentiate between different semens, as it gives a superimposable band for the original semen with its diluted sample, or for the extract of such dried stains with deionized water.
- The ratio of the androgens (T : DHT), i.e., testosterone (T), and its precursor dihydro-testosterone (DHT), is found to be specific for each and every individual.[14]

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Salivary stains on various substrates can be detected by the use of UV light, as they exhibit fluorescence. Detection of saliva by various chemical tests is based on the presence of amylase, thiocyanate ion, nitrite ion, alkaline phosphatase, etc. However, tests based on amylase are most commonly used. Qualitative amylase tests are used for screening purposes, whereas quantitative tests are reliable in confirming a salivary stain, since amylase is present in maximum concentration in saliva as compared to other body fluids. Several types of amylase isoenzymes have been identified exhibiting genetic variations and hence can be used for individualisation. Further individualization methods include ABO grouping, red cell enzyme and serum protein polymorphisms in secretors, and DNA fingerprinting.

Medicolegal Importance

1. Circumstantial evidence - The presence of saliva in cigarette butts, beedis, or sputum, at the scene of a crime can link the perpetrator to the crime.
2. Sexual offences - The presence of saliva in a bite mark (if present), can help to identify the offender.
3. Saliva is a non-invasive specimen which can be obtianed with ease from anyone to test for drugs of abuse.

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CHAPTER - 23

Torture and the Medical Profession

"Kill me! Kill me! But don't torture me with your murderous looks."
- Character in Samuel Samson's play **The Crossing**

Torture is a comparatively new entry in Forensic Medicine. It deals with medical, legal, and ethical issues in relation to the barbaric practice of torture in various situations. In the early days, in virtually any society, Western or Eastern, torture of alleged criminals, spies, and political prisoners was considered acceptable. Today, torture in any form is condemned by civilised societies, and is not acceptable in any situation. Yet, clandestinely, this is going on in some form or the other all over the world. Only the methods of torture have changed, and have become more "sophisticated", and consequently more "effective", and very difficult to detect medically. According to Amnesty International, the world-wide organisation that monitors human rights violations, torture is practiced even by governments in authority in 65 out of 144 countries, on those considered inimical to the state.

DEFINITION OF TORTURE

As per the **Tokyo Declaration of the World Medical Association in 1975, torture** is defined as *the deliberate or intentional, systematic wanton infliction of physical or mental suffering by one or more persons acting alone, or on the orders of any authority, to force another person to yield information, or to make a confession, or for any other reason.*

AIMS OF TORTURE

As per the definition given above, there is invariably some purpose or objective behind an act of torture. Some of the common reasons for torture are as follows -

1. **To obtain information:**
   It depends on the case and its gravity. If the person is suspected to have indulged in anti-national activity, or terrorist activity, governmental organizations are often involved in the infliction of torture. Such torture will usually continue till the desired information is obtained.

2. **To force a confession:**
   A person may be tortured in order to force him/her to confess to a crime. The victim is often coerced into signing a document of confession saying that he/she has committed the crime. Invariably the person eventually signs the confessional document under such threat and force, even though he/she may not have committed the crime, only to avoid further torture.

3. **To obtain testimony incriminating others:**
   Many a time, victims are forced into signing documents incriminating other people, whether they are involved or not, in order to build a case against them.

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4. **To take revenge:**
   Sometimes a victim is tortured merely to take revenge, which may be not only against the individual concerned, but also the family members of the victim, or even an entire community. For example, raping an enemy's wife, sister or daughter, kidnapping family members, especially small children, aged parents, etc.

5. **To spread terror in the community:**
   This is very common in dictatorial regimes. Anybody who dares to raise his/her voice against the regime, may be tortured mercilessly. If the victim dies, his body is often displayed to the community openly, with overt evidence of torture. This creates terror in the minds of the people, and nobody would then dare to rise up against the regime. The dictator thus manages to continue his/her dictatorial rule in the country.

6. **To destroy the personality:**
   There are always some people with leadership qualities in every community. These people are most likely to raise their voices against dictatorial rule, or oppression in the society. They are capable of mobilising people in the community against tyrants or dictators. Such persons are sometimes identified by the dictatorial regime, arrested, kidnapped, and tortured, so that they are transformed into a kind of "living dead". Their behaviour, thoughts, and feelings are suppressed, and they lose their self-confidence. There is often generation of feelings of hopelessness and worthlessness in the society.

METHODS OF TORTURE[1]

- Physical Torture
- Psychological Torture
- Sexual Torture

PHYSICAL TORTURE

Physical methods of torture are those which inflict pain, discomfort, and dysfunction in different parts of the body.

1. **Physical torture that causes extreme and excruciating pain:**
   - **Severe Beating** - is a common type of torture by using sticks, cables, whips, iron rods, etc.
   - **"Falanga" torture** - Beating on the soles of the feet. It is a systematic form of torture that is often used in our country. The person may become disabled for several years.
   - **Finger Torture** - Pencils or similar objects are placed in between two fingers and squeezed hard. Sometimes the fingers are twisted to cause severe pain.
   - **Cold Torture** - Victim is made to lie on an ice slab without clothing, or on a cold and damp floor.
   - **Heat Torture** - Victim is suspended upside down very near to fire, or cigarette burns are inflicted.
   - **Dental Torture** - Pulling out teeth by clips or forceps. Sometimes the victim is asked to chew hard on small stones, metal pieces, etc.

2. **Physical torture that causes fear of immediate death:**
   - **Electrical Torture** - Electric shock is applied to sensitive parts of the body such as nipples, genitals etc. Sometimes the victim is tied to a metallic bed and electricity is passed, causing shock all over the body.
   - **Sham Execution** - Victim is sometimes blind folded and asked to stand before a wall, and then threatened that a vehicle is going to hit him. The victim hears the sound of a vehicle very near to him, and coming towards him, causing fear and shock.

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3. **Physical torture that causes extreme exhaustion:**
   - **Physical Exhaustion** - The victim is asked to stand in hot sun on one leg for a long time. Or he may be asked to stretch out both his hands holding heavy bricks or stones.
   - **Forced Labour** - The victim is made to work very hard without food and water.

4. **Physical torture that causes disfiguration, mutilation or permanent disability:**
   - **"Telefono"** - In this method, simultaneous beating of both ears with the palms of the hands is done, causing rupture of tympanic membranes.
   - **Mutilation** - Chopping of ears, nose, fingers, etc.
   - **Disfiguration** - Throwing acids or corrosives over face and other parts of the body, causing severe burns leading to disfiguration.

MENTAL TORTURE

1. **Deprivation Techniques:**
   The victim is deprived of common and basic necessities, to induce depression.
   - **Sensory deprivation** - Victim may be blindfolded for a long time.
   - **Perceptual deprivation** - Making the victim get confused and disoriented, eg., frequent transfer of the victim from one place to another, after blindfolding him.
   - **Social deprivation** - Solitary confinement in a dark environment.
   - **Deprivation of basic needs** - Withholding food, water, toilet facilities, etc.

2. **Coercion Techniques:**
   These methods of torture involve the compelling of a victim to perform certain acts, or witness certain traumatic acts, which can cause mental anguish, e.g.,
   - signing of false statements.
   - coercion to commit blasphemous acts.
   - coercion to witness torture of other victims.
   - coercion to torture other victims.

3. **Communications Techniques:**
   The victim is mentally tortured by exposure to a variety of confusing, contradictory, or false communications, such as
   - **Misinformation** - Giving false information to victim regarding tragedy involving close relatives such as wife and children, which causes mental anguish.

SEXUAL TORTURE

This method of torture inflicts physical and psychological damage on the victim,
- **Sexual torture using instruments** – Infliction of injuries to private parts of both male and female victims, forcible introduction of rods, bottles, or batons in the rectum or vagina, mutilation of breasts or genital organs, etc.
- **Sexual torture without the use of instruments** - The victim may be forced to undress in front of others, or raped in front of others, or photographed in humiliating positions or situations, or sexually tortured by trained animals.

CONSEQUENCES OF TORTURE[2]

PHYSICAL CONSEQUENCES

Early Consequences

Injuries in the form of abrasions, bruises, haematomas, lacerations, incised wounds, penetrating wounds, dislocations, fractures, etc.

Late consequences

Infections, scars, malunited bones, disfiguration of the face, impairment of hearing, impairment of vision, vertigo, etc.

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PSYCHOLOGICAL CONSEQUENCES

Anxiety, depression, sleep disturbances, alcohol/drug abuse, seizures, low self esteem, stress disorders, etc.

SOCIAL CONSEQUENCES

Social stigma, employment problems, negativity in social activities, etc.

TREATMENT OF TORTURE VICTIMS[3]

As discussed earlier, torture has three dimensions - physical, psychological, and social. It is very difficult to assess which is more important, and which is less, in a particular individual. Treatment may not be effective if it is focussed on only one dimension. Doctors, psychologists, social workers, physiotherapists, nurses, and legal experts must work together in close collaboration, for maximum efficacy.

Principles of Treatment [4]

Torture victims and their families are a special category of clients, because they have experienced an unusual (and often brutal) trauma at the hands of other human beings. Therefore, while dealing with torture victims and their families, the following principles need to be kept in mind -

- Developing a good rapport with the victim.
- Empathising with the victim and his/her family.
- Avoiding situations or objects that remind the victim of the torture event.
- Punctuality in keeping appointments with the victim, and his/her family, should be strictly maintained.

MEDICO-LEGAL AND ETHICAL ASPECTS OF TORTURE[5]

- Under the **Hippocratic Oath**, doctors are prohibited from using their professional knowledge to harm their patients, which was reconfirmed in the **Geneva Declaration** passed by the World Medical Association. However, in spite of this, it is sadly true that doctors sometimes involve themselves directly or indirectly in the perpetration of torture, especially military doctors, police and prison doctors, and forensic experts.
- The **World Medical Assembly** (WMA), in **Tokyo (1975)**, adopted a special resolution in regard to this. The following are the salient points of the **Tokyo Declaration**:
  1. A doctor shall not countenance, condone or participate in the practice of torture or other forms of cruel, inhuman, or degrading procedures.
  2. A doctor shall not provide any premises, instruments, substances, or knowledge to facilitate the practice of torture.
  3. Doctors should not be present during any procedure involving cruel or inhuman activities.
  4. A doctor must have complete clinical independence in deciding upon the care of a person for whom he or she is medically responsible.
  5. Doctors shall in all circumstances consider it their bounden duty to alleviate the distress of their fellowmen, and no motive, whether personal, collective, or political, shall prevail against this higher purpose.
  6. The World Medical Association will support and encourage the international community, the national medical associations, and fellow professional colleagues, to support a doctor and his or her family, in the face of threats or reprisal resulting from a refusal to condone the use of torture or other forms of cruel, inhuman, or degrading treatment.
- Victims of torture may either suffer from injuries, or may die because of injuries. Such cases should be examined carefully and thoroughly, and reported to the police or magistrate, and

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treated appropriately. Systematic and meticulous examination will help in establishing or disproving a charge of torture. **Sections 330** and **331** of the **IPC** deal with the issue of voluntarily causing hurt and grevious hurt for the purpose of extorting confession or any information.

- The government of India has enacted the **Human Rights Act, 1993**, which monitors the violation of any of the rights of the individuals of this country.

Box 23.1 Outlines some actual case histories of victims of torture.

Box 23.1 Case Histories in Torture

Case 1

Mr.P. is a 55 year old man from mid-eastern Nepal. He is married and lives with his wife and two children. Mr.P. was a political activist, and participated in armed activities against the government. One day, he was arrested along with some of his friends, charged with anti-government activities, and kept in police custody for several days.

He was handcuffed and kept in fetters while in custody. During this period, he was repeatedly whipped and beaten with sticks on the thighs, shoulders, and the soles of his feet. He was boxed on both ears, and chilly powder was forced into his nostrils. His fingers and toes were burnt with smouldering cigarettes. He became unconscious several times when electric shock was applied to his body. Threats on his life, and the lives of his family members were repeatedly made. He was humiliated and kept in total isolation. He was not allowed to go to the toilet and was held incommunicado throughout his period in custody. He was given contaminated water to drink and stale food mixed with stones and glass pieces to eat. Later he was transferred to a prison where he was kept in detention for 3 years.

Mr.P. was referred to the Centre for Victims of Torture (CVICT), Nepal, by one of his friends. At the time of his examination, he complained of diminished vision, joint and muscle pain, low back pain, and burning and pricking sensations over the soles. He found it extremely difficult to lead a normal life. At the CVICT, his complaints were thoroughly investigated, and he was put on analgesic therapy, psychotherapy, and physiotherapy. Today his condition is much better, and he is now leading a normal life with his family.

Case 2

Mr. B, a 32 year old university student, was arrested in 1980 from his residence on the charge of being an active member of a political party. He spent 15 days in police custody and 3 months in jail. During this period, he was kept in a cold storage unit for one night. Unable to bear the cold, he became unconscious after two hours. He was then subjected to electric torture. He was kept naked throughout the process. After his release, Mr.B was arrested a second time, from the University Campus. The police tied his neck with a rope and made him crawl to the police station. In custody, he was kicked all over the body with boots till his body was soaked with blood. He was later transferred to a prison where he was beaten every night. Police repeatedly threatened to kill him, and told him that if he changed his political ideology they would stop torturing him.

Upon his release after several weeks, Mr. B was unable to sleep at night and had nightmares of torture practically every night. He became mentally unstable and had to be taken to a mental hospital many times. He frequently quarreled with his family members. He did not like to dress properly, and talked to himself most of the time. He used to get tired easily and had difficulty in thinking clearly and making

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decisions.He had problems with his concentration, and experienced feelings of hopelessness. He felt himself defeated in every aspect of life - political, social and family life. On examination by a psychiatrist at the Mental Hospital, he was diagnosed as a case of schizophrenia. Treatment for schizophrenia was begun, but he showed no improvement.

Mr. B was referred to the CVICT on April 12, 1991, where he was seen by the consultant psychiatrist who diagnosed his condition as one of *post-traumatic stress disorder.* He was given appropriate treatment, and was fully informed about the therapeutic principles of the centre. The CVICT carried out an integrated program of physiotherapy, social counseling, psychotherapy and pharmacotherapy. Today, Mr.B is able to adjust himself in society, and has regained his concentration and memory power. He is still under medication, but manages to lead a fairly normal life.

Case 3

Ms. M, a 26 year old girl, left her village for the city, where she was employed in one of the carpet factories. At 7.00 p.m. on December 1, 1993, while she was preparing her evening meal at home, two policemen knocked on her door and informed her that she had been called to the police station by the Sub-Inspector. Ms. M refused to go with them as she had not committed any crime. On failing to convince Ms.M, the police went back, but returned later in the night and forcibly took her to the police station, where the Sub Inspector asked her why she had refused to come the first time. He then caught her by her hair and dragged her into a room. He slapped her several times and randomly beat her all over the body with stick and rifle butt. He abused her and raped her. Three other police men also raped her. She then fell unconscious.

The next day, some friends of Ms. M began enquiring about her whereabouts and surrounded the police station. Ms. M was taken to a hospital in a police van. No visitors were allowed to see her or talk to her. From the hospital, she was again taken back to police custody. This time, she was verbally humiliated and charged falsely with prostitution.

After three days, two policemen took her to another town. On the way, she spent several days in various police stations. On different occasions, she was stripped completely, and raped repeatedly. After 35 days, she finally reached home. The police threatened to kill her if she took any legal action. Five months after the event, she was brought to the CVICT for treatment. She was referred to the centre by an organisation and a human rights lawyer. At the time of referral, she was suffering from physical and psychological problems. She complained of irregular, excessive menstrual bleeding, and generalised bodyache. She re-experienced her traumatic events in her mind constantly, and felt terrified at the sight of police and army personnel. She suffered from severe insomnia.

At the CVICT, she was assessed thoroughly by a joint team comprising of a psychiatrist, a social worker, and a physiotherapist. She was also seen by a gynaecologist and treatment begun immediately. She was placed on regular counselling, and antidepressant treatment. Later on, desensitization was carried out by exposing her to the police and army personnel in a graded manner. At first she was very disturbed, but after continuous treatment over a period of several months, she gradually developed self-esteem, and finally went back home with her family members. She was diagnosed as a case of *post-traumatic stress disorder.* At present, she is with her parents and leading a functional life. She has been successfully running a small restaurant in her village with capital provided by the CVICT.

Adapted from **Shrestha NM, Sharma B.** *Torture and Torture Victims : A Manual for Medical Professionals.* **1995.** Centre for Victims of Torture, Nepal.

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SECTION - B

Toxicology

"Caution is advised while decontaminating a victim of poisoning. Overenthusiasm may result in eliminating the patient, not the poison!".

- John Morrison

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CHAPTER - 1

General Toxicology

The causes of poisoning are many - civilian and industrial, accidental and deliberate. It has been estimated that some form of poison directly or indirectly is responsible for more than 1 million illnesses world-wide annually, and this figure could be just the tip of the iceberg since most cases of poisoning actually go unreported, especially in third world countries. The problem is getting worse with time as newer drugs and chemicals are developed in vast numbers. Today there are more than 9 million natural and synthetic chemicals, and the list keeps growing inexorably. However, less than 3000 of these cause more than 95% of the reported cases of poisoning. The commonest agents in India appear to be **pesticides** (organophosphates, carbamates, chlorinated hydrocarbons, and pyrethroids), **sedative drugs, chemicals** (corrosive acids and copper sulfate), **alcohol, plant toxins** (datura, oleander, strychnos, and gastro-intestinal irritants such as castor, croton, calotropis, etc.), **and household poisons** (mostly cleaning agents). Of late, **aluminium phosphide** has begun to emerge as a major player in the toxicological field, particularly in some northern Indian states. Among children the common culprits include **kerosene, household chemicals, drugs, pesticides, and garden plants.**[1]

POISON INFORMATION CENTRES

Arising out of a growing concern over the burgeoning incidence of poisoning world wide, coupled with a lack of public awareness about its seriousness, **Poisons Information Services** made their first appearance in the Netherlands in 1949. In 1961, a telephone answering service was introduced in Leeds, England, which gave information to medical practitioners and others about the poisonous properties of a variety of household, agricultural, and therapeutic substances. On 2 September 1963, a National Poisons Information Service was established at Guy's Hospital, London. The same year, the Illinois Chapter of the American Academy of Paediatrics opened an information centre in Chicago, USA. Since then, all around the world similar centres have sprung up, performing the invaluable functions of generating public awareness on poisoning, and imparting much needed toxicological diagnostic and therapeutic assistance to doctors. Today there are 75 such certified centres in the USA alone, providing almost any information within a matter of seconds through the use of an intricate, computerised information resource system (POISINDEX) on more than 800,000 poisonous products.[2] India made a belated foray with the establishment of the **National Poisons Information Centre** at the **All India Institute of Medical Sciences, New Delhi** in December, 1994. The World Health Organisation has released its computer soft-

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ware on poisons (INTOX) for use by the centre. A second centre was subsequently opened at the **National Institute of Occupational Health, Ahmedabad**. Efforts are on now to establish similar centres in other parts of the country.[3]

In Ireland, Spain, and the Netherlands, the poison centres are integrated with diagnostic laboratories unlike those in the USA which function independently, while in England, France, and Germany, the centres work in conjunction with acute poisoning treatment units as well as diagnostic laboratories. Among countries of the Asia Pacific, Australia, New Zealand, Indonesia, Singapore, Philippines, Hong Kong, Pakistan, Sri Lanka, Iran, and Turkey have established poison information centres, apart from India. Among Latin American countries, Brazil has 30 centres while Uruguay has one well established centre. Very few countries in the African continent with the exception of South Africa, Zimbabwe, and Kenya have such centres. Countries such as Russia, China, and East European countries are also at present deficient, but are making efforts at establishing them.

Poison centres provide immediate, round the clock toxicity assessment and treatment recommendation over the telephone for all kinds of poisoning situations affecting people of all ages, including ingestion of household products, overdose of therapeutic medication, illegal foreign and veterinary drugs, chemical exposures on the job or elsewhere, hazardous material spills, bites of snakes, spider and other venomous creatures, and plant and mushroom poisoning. When a call about a poisoning is received, the poison information specialist obtains a history from the caller, assesses the severity of the poisoning, provides treatment recommendations, and refers the patient for further medical attention when necessary. Referrals to health care facilities when made are later followed up with phone calls to assess progress, and provide additional recommendations until any medical problems related to the poisoning are resolved. Information from the beginning of the call to the final outcome are noted on preformatted case sheets, and quantifiable data is filled in by darkening respective bubbles on the sheet. The data generated is periodically analysed by the centre and is also monitored for quality assurance of the information specialists.

A typical regional poison centre is located at a university medical college or hospital, serves a population of approximately 4 million people, and handles about 35,000 human exposure cases every year. It is staffed by a **physician** (Medical Director), a **pharmacist** (Technical Director), an **administrator**, an average of 10 **poison information specialists**, a **secretary**, and a **staff member devoted to community education.**

Poison information specialists are consulted daily by parents, grand parents, child care providers, pharmacists, ambulances, responders to hazardous material exposure incidents, nurses, and physicians in every area of medical practice from neonatology to geriatrics, clinical medicine to occupational medicine, and family practice to intensive care. Nearly 75% of poisonings reported to poison centres are managed entirely by telephone consultations without further necessity of additional costs for the health care system.

MORTALITY FROM POISONING

This varies from country to country depending on the kind of poisons encountered, the extent of awareness about poisoning, the availability of treatment facilities, and presence or absence of qualified personnel. While in developed countries the rate of mortality from poisoning is as low as 1 to 2%, in India it varies from a shocking 15 to 35%.[4] Children under 15 years of age account for most cases of accidental poisoning, but fortunately they are associated with relatively low mortality.

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On the other hand, most suicidal exposures are seen in individuals over 15 years of age but are associated with high mortality.

In poisoning cases, the attending physician is often asked to comment on the prognosis of the victim's condition. Unfortunately in cases of serious poisoning, it is very difficult to predict the outcome. There are many reasons for this. In a substantial number of cases, the doctor is unaware of the exact nature of the poison consumed; in others, the victim may have ingested several kinds of drugs simultaneously. Even in those cases where the exact identity and dose of a single ingested poison is known, the doctor may not have a clear idea as to its toxicity. In order to ameliorate the situation to some extent and help physicians have some idea as to the hazardous nature of various poisons, a system of "**toxicity rating**" has been evolved for common poisons. The higher the toxicity rating for a particular substance (over a range from 1 to 6), the greater its potency (**Table 1.1**). The rating is based on mortality, and is applicable only to the acute toxicity of a single dose taken orally. In the case of commercial products where various combinations of poisonous substances may have been used, one has to derive an estimate of the toxicity rating in totality, taking into consideration all the components put together with particular reference to individual concentrations.

Table 1.1 Toxicity Rating

Usual Fatal Dose	Rating
Less than 5 mg/kg	6 (Super Toxic)
5 to 50 mg/kg	5 (Extremely Toxic)
51 to 500 mg/kg	4 (Very Toxic)
501 mg/kg - 5 gm/kg	3 (Moderately Toxic)
5.1 gm/kg - 15 gm/kg	2 (Slightly Toxic)
More than 15 gm/kg	1 (Practically Non Toxic)

To assess and rate the toxicity of a drug, the **Usual Fatal Dose** (UFD) is taken into consideration which is derived from animal experimental data and statistics of human poisoning. The UFD is based on the **Minimum Lethal Dose** (MLD) which is usually indicative of the lethal dose that is fatal to 50% of animals (LD 50). While the UFD of virtually every poison/drug finds mention in this book under the relevant section, **Table 1.2** serves as a quick reference source for common agents.

DIAGNOSTIC CONSIDERATIONS

A poisoning case can present to a doctor or hospital in any one of a number of ways. Broadly, there are four types of presentation :

1. **Fulminant:** Produced by a massive dose. Death occurs very rapidly, sometimes without preceding symptoms, the patient appearing to collapse suddenly.
2. **Acute:** Produced by a single dose or several small doses taken in a short period. Onset of symptoms is abrupt.
3. **Chronic:** Produced by small doses taken over a long period. Onset is insidious.
4. **Subacute:** Characterised by a mixture of features of acute and chronic poisoning.

In most cases, the poisoned patient presents with one or more of the following non-specific features:

- Impairment of consciousness
- Respiratory/Cardiovascular depression
- Dehydration due to vomiting/diarrhoea
- Hypothermia
- Convulsions
- Cardiac arrhythmias

Several drugs/poisons affect the pupils of the eyes producing either miosis or mydriasis. A few produce nystagmus. These have been laid out in **Table 1.3**.

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Table 1.2 Usual Fatal Dose of Common Toxic Agents

Acetyl salicylic acid (Aspirin)	15 to 20 gm	Cyanide (salt)	200 to 300 mg	Methanol	60 to 250 ml
Acids (Mineral)	10 to 15 ml	Datura	50 to 75 seeds	Morphine	200 mg
Aconite (Root)	1 gm	DDT	15 to 30 gm	Nicotine	60 mg
Aluminium Phosphide	3 gm	Diazinon	1 gm	Oleander	5 to 15 leaves or 15 gm root
Arsenic trioxide	250 mg	Ethanol	5 to 8 gm/kg	Opium	500 mg
Atropine	10 mg	Ethylene glycol	100 ml	Organochlorines (except Lindane and DDT)	2 to 6 gm
Long - acting Barbiturate	3 gm	Formaldehyde	30 to 60 ml	Oxalic acid	15 to 20 gm
Short - acting Barbiturate	1 to 2 gm	Heroin	50 mg	Paracetamol	12 to 20 gm
Benzene	15 to 20 ml	Iron	200 mg/kg	Parathion	100 mg
Carbolic acid (Phenol)	20 ml	Isopropanol	200 to 250 ml	Phosphorus	60 to 120 mg
Castor	5 to 10 seeds	Lead acetate	10 gm	Strychnine	50 to 100 mg
Cocaine	1 to 2 gm	Lindane	15 to 30 gm	TEPP	100 mg
Copper sulfate	30 gm	Malathion	1 gm	Thallium (salt)	1 gm
Curare	60 mg	Mercuric chloride	1 to 2 gm		

Some poisons have distinctive odours which may be perceived in the vicinity of a poisoned patient, especially in the breath. Some important examples are mentioned in **Table 1.4**

GENERAL MANAGEMENT OF POISONING

1. **Stabilization:** The initial survey should always be directed at the assessment and correction of life threatening problems, if present. Attention must be paid to the airway, breathing, circulation, and depression of the CNS (the **ABCD** of resuscitation).
2. **Evaluation:** If the patient is not in crisis, i.e., he is alert with normal speech and pulse, proceed to a complete, thorough, and systematic examination. As far as treatment is concerned, the emphasis should be on basic supportive measures.
3. **Decontamination:** This is with reference to skin / eye decontamination, gut evacuation and administration of activated charcoal.

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Table 1.3 Drugs/Poisons Producing Pupillary Changes

Miosis	Mydriasis	Nystagmus
Barbiturates	Alcohol (constricted in coma)	Alcohol
Benzodiazepines	Amphetamines	Barbiturates
Caffeine	Antihistamines	Carbamazepine
Carbamates	Carbon monoxide	Phencyclidine
Carbolic acid (Phenol)	Cocaine	Phenytoin
Clonidine	Cyanide	
Methyl dopa	Datura (Atropine)	
Nicotine	Ephedrine	
Opiates		
Organophosphates		
Parasympathomimetics		

Table 1.4 Diagnostic Odours

Odour	Substance
Acetone (apple – like)	Chloroform, ethanol, isopropanol, lacquer
Acrid (pear – like)	Chloral hydrate, paraldehyde
Bitter almond	Cyanide
Burnt rope	Marijuana (Cannabis)
Coal gas	Carbon monoxide (CO)
Disinfectant (hospital odour)	Carbolic acid, creosote
Garlicky	Arsenic, dimethylsulfoxide, organophosphates, phosphorus, selenium, tellurium, thallium
Mothballs	Camphor, naphthalene
Musty (fishy)	Aluminium phosphide, zinc phosphide
Rotten egg	Carbon di sulfide, disulfiram, hydrogen sulfide, mercaptans, N-acetylcysteine
Shoe polish	Nitrobenzene
Vinegar	Acetic acid
Wintergreen	Methyl salicylate

4. **Poison Elimination:** Depending on the situation, this can be accomplished by diuresis, peritoneal dialysis, haemodialysis, haemoperfusion, etc.

5. **Antidote Administration:** Unfortunately, antidotes are available for less than 5 % of poisonings.

6. **Nursing and Psychiatric Care:** General nursing care is especially important in comatose patients, and those who have been incapacitated by the poison. Since some cases of poisoning leave behind persisting sequelae, adequate follow-up for a period of time may be necessary. Psychiatric intervention is frequently essential in suicidal overdose.

Stabilization & Evaluation

The immediate need for assisted ventilation has to be assessed clinically, but the efficiency of ventilation can only be gauged by measuring the blood gases. Retention of carbon dioxide ($PaCO_2 > 45$ mm Hg or 6 Kpa), and hypoxia ($PaO_2 < 70$ mm Hg or 9.3 Kpa) inspite of oxygen being given by a facemask are indications for assisted ventilation. A simple method of assessing respiratory status consists of determining the minute volume by a Wright spirometer. If this is more than 4 litres / min., there is no need for ventilation. However it must be borne in mind that ventilatory function can fluctuate and may deteriorate suddenly.

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Table 1.6 Drugs associated with disturbances in pulse rate and blood pressure

Tachycardia & Normotension	Tachycardia & Hypotension	Tachycardia & Hypertension	Bradycardia & Hypotension	Bradycardia & Hypertension
Antihistamines, caffeine, cannabis, lomotil (atropine & diphenoxylate), thyroxine	Carbon monoxide, cyanide, phenothiazines, theophylline	Amphetamines, cocaine, phencyclidine, phenyl propanolamine	Clonidine, levodopa, MAOIs, organo-phosphates, opiates, tricyclic antidepressants	Phenylpropanolamine

Table 1.5 lists some substances which are known to cause respiratory depression. Some drugs stimulate the respiratory centre : amphetamines, atropine, cocaine, and salicylates.

Table 1.5 Toxic Respiratory Depression

Failure of Respiratory Centre	Failure of Respiratory Muscles
Antidepressants	Neuromuscular blocking agents
Antipsychotics	Nicotine
Ethanol	Organophosphates
Opiates	Shell fish poisoning
Sedatives	Snake bite (Cobra)
Strychnine	

Several drugs produce changes in pulse rate and blood pressure (**Table 1.6**)

There are numerous causes for coma of which one of the most important is acute poisoning. A number of substances can induce coma (**Refer Table 1.7**)

Hoffman and **Goldfrank** reviewed extensive data from 1966 to 1994 on the management of poisoned comatose patients and came to the clinically rational conclusion that in every case where the identity of the poison was not known, the following three antidotes (called the **Coma Cocktail**) must be administered.[5]

1. Dextrose – 100 ml of 50% soln.
2. Thiamine (Vitamin B1)–100 mg
3. Naloxone – 2 mg

All given i.v.

For a child, thiamine is not recommended, and the dextrose should be more dilute (20 to 25 %). If i.v., access is a problem, naloxone can be given intramuscularly or even endotracheally.

The rationale for the **coma cocktail** is that a significant proportion of poisoned comatose patients in whom the identity of the poison is unknown, comprise cases of overdose from opiates, alcohol, and hypoglycaemic agents. Even if a particular case is not due to any of these causes, administration of these antidotes is relatively harmless. Physostigmine must not be a part of the cocktail, since even though coma from anticholinergic excess is also relatively common, physostigmine which is the antidote for it, produces serious side effects such as seizures. It should be administered (2 mg i.v for an adult, and 0.5 mg in the child) only if there are unequivocal signs of anticholinergic excess.

In the case of metabolic acidosis, it is necessary to calculate the **anion gap**. The anion gap is calculated as follows: $(Na^+ + K^+) - (HCO3^- + Cl^-)$

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Table 1.7 Toxic Causes of Coma

1. Hypoxia	
A. *Displacement of oxygen in blood and tissues*	B. *Displacement of oxygen in the atmosphere*
Carbon monoxide	Carbon dioxide
Agents causing methaemoglobinaemia	Butane
Acetanilid	Propane
Aniline dyes	Methane
Chlorates	Dinitrophenol
Nitrites	
2. Acidosis	
Ethylene glycol	
Isopropanol	
Methanol	
Paraldehyde	
Salicylates	
3. Depression of CNS	
Alcohols: ethanol, methanol, isopropanol	Benzodiazepines
Anticonvulsants	Bromides
Antidepressants	Chloral hydrate
Antihistamines	Opiates
Barbiturates	Phenothiazines
4. Hypoglycaemia	
Ethanol	
Hypoglycaemic drugs	
Insulin	
Isoniazid	
Salicylates	
5. Enzyme inhibition	
Cyanide	
Heavy metals	
Organophosphates	
6. Post-ictal	
Amphetamines	
Cocaine	
Chlorinated hydrocarbons	
Hallucinogens	
Withdrawal from alcohol, sedatives, etc.	
7. Other causes	
Food poisoning : Botulism, mushrooms, shellfish, etc.,	
Snake bite	

Modified from **Ellenhorn MJ.** *Medical Toxicology : Diagnosis and Treatment of Human Poisoning.* 2nd Edn, *1997.* Williams and Wilkins, Baltimore, USA, p18.

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Table 1.8 Causes of Acid - Base Disorders

Type of disorder	Causes	
Acute respiratory alkalosis	Anxiety, hypoxia, lung diseases, CNS disease, pregnancy, sepsis, hepatic encephalopathy, drugs - salicylates, catecholamines, progesterone, etc.	
Acute respiratory acidosis	CNS depression, acute airway obstruction, neuromuscular disorders (myopathies, neuropathies) severe pneumonia or pulmonary oedema, impaired lung motion (haemothorax, pneumothorax)	
Chronic respiratory acidosis with metabolic compensation	Chronic lung disease, chronic neuromuscular disorders, chronic respiratory centre depression	
Metabolic alkalosis	**Normal or high urinary chloride** Cushing's disease, Conn's syndrome, steroid administration, diuretic administration, alkali overdose	**Low urinary chloride** Vomiting, past use of diuretics, post-hypercapnia
Metabolic acidosis	**Non - Gap** Diarrhoea, renal tubular acidosis, carbonic anhydrase inhibitors, aldosterone inhib--tors, post-hypocapnia, brom--ism, iodism, and secondary to hyperkalemia, hypercalcemia	**Gap*** Methanol Uremia Diabetes Paraldehyde, phenformin Idiopathic lactic acidosis, iron, isoniazid Ethanol, ethylene glycol Salicylates, solvents, starvation

**The important causes can be remembered by the acronym - MUDPIES.*

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Normally this translates as
140 - (24 + 104) = 12 mmol / L (Range : 12 to 16 mmol / L)

If the anion gap is greater than 20 mmol / L, a metabolic acidosis is present regardless of the pH or serum bicarbonate concentration. Several poisons are associated with increased anion gap (**Gap acidosis**), while others do not alter it (**Non-Gap acidosis**). The common causes for the various acid-base disorders are mentioned in **Table 1.8**

Decontamination

The various methods of poison removal from the gastro-intestinal tract include

1. Emesis
2. Gastric lavage
3. Catharsis
4. Activated charcoal
5. Whole bowel irrigation

1. **Emesis:** The only recommended method of inducing a poisoned patient to vomit is administration of **syrup of ipecacuanha** (or **ipecac**) and ironically it is not easy to procure it in India

   Source - Root of a small shrub (*Cephaelis ipecacuanha* and *C. acuminata*) which grows well in W.Bengal.

   Active principles - Cephaeline, emetine, and traces of psychotrine.

   Indications - Conscious and alert poisoned patient who has ingested a poison not more than 4 to 6 hours earlier.

   Mode of action
   a. Local activation of peripheral sensory receptors in the gastrointestinal tract.
   b. Central stimulation of the chemoreceptor trigger zone with subsequent activation of the central vomiting centre.

   Dose: 30ml (adult), or 15ml (child), followed by 8 to 16 ounces i.e., 250 to 500 ml approximately, of water. The patient should be sitting up. If vomiting does not occur with in 30 minutes, repeat the same dose once more. If there is still no effect, perform stomach wash to remove not only the ingested poison but also the ipecac consumed! However the therapeutic doses of ipecac recommended above are not really harmful.

   Contra-indications: *Relative*:
   - Very young (less than 1 year), or very old patient
   - Pregnancy
   - Heart disease
   - Bleeding diathesis
   - Ingestion of cardiotoxic poison
   - Time lapse of more than 6 to 8 hours

   Absolute
   - Convulsions, or ingestion of a convulsant poison
   - Impaired gag reflex
   - Coma
   - Foreign body ingestion
   - Corrosive ingestion
   - Ingestion of petroleum distillates, or those drugs which cause altered mental status (phenothiazines, antihistamines, opiates, ethanol, benzodiazepines, tricyclics)
   - All poisons which are themselves emetic in nature

   Complications
   - Cardiotoxicity (bradycardia, atrial fibrillation, myocarditis)
   - Aspiration pneumonia
   - Oesophageal mucosal or Mallory Weiss tears (due to protracted vomiting)

2. **Gastric Lavage (Stomach Wash):** Indications- Gastric lavage is recommended

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mainly for patients who have ingested a life-threatening dose, or who exhibit significant morbidity and present within 1 to 2 hours of ingestion. Lavage beyond this period may be appropriate only in the presence of gastric concretions, delayed gastric emptying, or sustained release preparations. Some authorities still recommend lavage upto 6 to 12 hours post-ingestion in the case of salicylates, tricyclics, carbamazepine, and barbiturates.

Precautions

a. Never undertake lavage in a patient who has ingested a non-toxic agent, or a non-toxic amount of a toxic agent.

b. Never use lavage as a deterrent to subsequent ingestions. Such a notion is barbaric, besides being incorrect.

Contra-indications

Relative: Haemorrhagic diathesis,oesophageal varices, recent surgery, advanced pregnancy, ingestion of alkali, coma.

Absolute: Marked hypothermia, prior significant vomiting, unprotected airway in coma, and ingestion of acid or convulsant or petroleum distillate, and sharp substances.

Procedure

1. Explain the exact procedure to the patient and obtain his consent. If refused, it is better not to undertake lavage because it will amount to an assault, besides increasing the risk of complications due to active non-cooperation.
2. Endotracheal intubation must be done prior to lavage in the comatose patient.
3. Place the patient head down on his left lateral side (20° tilt on the table).
4. Mark the length of tube to be inserted (50 cm for an adult, 25 cm for a child.).
5. The ideal tube for lavage is the **lavacuator** (clear plastic or gastric hose).

   In India however, the **Ewald tube** is most often used, which is a soft rubber tube with a funnel at one end, (**Fig 1.1**). Whatever tube is used, make sure that the inner diameter corresponds to at least 36 to 40 French size. A nasogastric tube used for gastric aspiration is inadequate and should never be used. In a child, the diameter should be at least 22 to 28 French, (**Ryle's tube** may be sufficient).
6. The preferred route of insertion is oral. Passing the tube nasally can damage the nasal mucosa considerably and lead to severe epistaxis.
7. Lubricate the inserting end of the tube with vaseline or glycerine, and pass it to the desired extent. Use a mouth gag so that the patient will not bite on the tube.
8. Once the tube has been inserted, its position should be checked either by air insufflation while listening over the stomach, or by aspiration with pH testing of the aspirate, (acidic if properly positioned).
9. Lavage is carried out using small aliquots (quantities) of liquid. In an adult, 200 to 300 ml aliquots of warm (38° C) saline or plain water are used. In a child, 10 to 15 ml/kg body weight of warm saline is used each time. Water should preferably be avoided in young children because of the risk of inducing hyponatremia and water intoxication. It is advisable to hold back the first aliquot of washing for chemical analysis.

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10. In certain specific types of poisoning, special solutions may be used in place of water or saline (**Table 1.9**).

Table 1.9 Solutions for Gastric Lavage

Poison	Solution
Most poisons (known or unknown)	Water or saline
Oxidizable poisons (alkaloids, salicylates, etc)	Potassium permanganate (1 : 5000 or 1 : 10000)
Cyanides	Sodium thiosulfate (25%)
Carbolic acid (phenols)	Castor oil and warm water (1 : 2)
Oxalates	Calcium gluconate
Iron	Desferrioxamine (2 gm in 1 litre of water)

11. Lavage should be continued until no further particulate matter is seen, and the efferent lavage solution is clear. At the end of lavage, pour a slurry of activated charcoal in water (1 gm/kg), and an appropriate dose of an ionic cathartic into the stomach, and then remove the tube.

Complications

1. Aspiration pneumonia.
2. Laryngospasm.
3. Sinus bradycardia and ST elevation on the ECG.
4. Perforation of stomach or oesophagus (rare).

3. **Catharsis** The two main groups of cathartics used in toxicology include

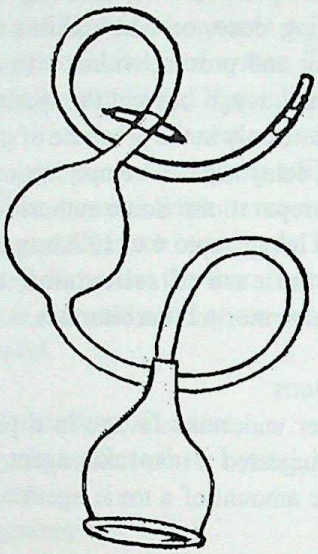

Fig 1.1 Stomach Tube

1. **Ionic or Saline:** These cathartics alter physico-chemical forces within the intestinal lumen leading to osmotic retention of fluid which activates motility reflexes and enhances expulsion. However, excessive doses of magnesium-based cathartics can lead to hypermagnesemia which is a serious complication.

   The doses of recommended cathartics are as follows:
   - Magnesium citrate - 4 ml/kg
   - Magnesium sulfate - 30 gm (250 mg/kg in a child)
   - Sodium sulfate - 30 gm (250 mg/kg in a child)

2. **Saccharides:** Sorbitol (D-glucitol) is the cathartic of choice in adults because of better efficacy than saline cathartics, but must not be used as far as possible in young children owing to risk of fluid and electrolyte imbalance (especially hypernatremia). It occurs naturally in many ripe fruits

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and is prepared industrially from glucose, retaining about 60% of its sweetness. Sorbitol is used as a sweetener in some medicinal syrups, and the danger of complications is enhanced in overdose with such medications when sorbitol is used as a cathartic during treatment.

Dose of sorbitol - 50 ml of 70% solution (adult)

Efficacy of catharsis - While cathartics do reduce the transit time of drugs in the gastrointestinal tract, there is no real evidence that it reduces morbidity or mortality in cases of poisoning.[6]

4. **Activated Charcoal:** Activated charcoal is a fine, black, odourless, tasteless powder made from burning wood, coconut shell, bone, sucrose, or rice starch, followed by treatment with an activating agent (steam, carbon dioxide, etc). The resulting particles are extremely small, but have an extremely large surface ar-ea. Each gram of activated charcoal works out to a surface area of 1000 square metres. Recently in the USA, a new **superactivated charcoal** has been introduced in the market with a surface area nearly double the current formulations.

Mode of action - Decreases the *absorption* of various poisons by *adsorbing* them on to its surface. Activated charcoal is effective to varying extent, depending on the nature of substance ingested (**Table 1.10**).

Dose - 1 gm/kg body weight (usually 50 to 100 gm in an adult, 10 to 30 gm in a child).

Procedure - Add four to eight times the quantity of water to the calculated dose of activated charcoal, and mix to produce a slurry or suspension. This is administered to the patient after emesis or lavage, or as sole intervention. The slurry should be shaken well before administration.

Disadvantages
1. Unpleasant taste.
2. Provocation of vomiting.

Table 1.10 Adsorption of toxins to activated charcoal

Well Adsorbed		Moderately Adsorbed	Poorly Adsorbed
Aflatoxins	Cimetidine	Antidiabetic drugs	Alcohols
Amphetamines	Dapsone	Kerosene	Carbamates
Antidepressants	Digitalis	Paracetamol	Corrosives
Antiepileptics	NSAIDs	Phenol	Cyanide
Antihistamines	Opiates	Salicylates	Ethylene glycol
Atropine	Phenothiazines		Heavy metals
Barbiturates	Quinine, Quinidine		Hydrocarbons
Benzodiazepines	Strychnine		Organophosphates
Beta blockers	Tetracycline		
Chloroquine	Theophylline		

Modified from ***Neuvonen PJ, Olkolla KT. Oral activated charcoal in the treatment of intoxication: Role of single and repeated dose.*** Med Toxicol **1998 ; 3 : 33 – 58.**

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3. Constipation/diarrhoea.
4. Pulmonary aspiration.
5. Intestinal obstruction (especially with multiple-dose activated charcoal).

Contraindications

1. Absent bowel sounds or proven ileus.
2. Small bowel obstruction.
3. Caustic ingestion.
4. Ingestion of petroleum distillates.

Elimination:

The various methods of eliminating absorbed poisons from the body include the following:

1. Forced Diuresis
2. Extracorporeal Techniques
   a) Haemodialysis
   b) Haemoperfusion
   c) Peritoneal dialysis
   d) Haemofiltration
   e) Plasmapheresis
   f) Plasma perfusion
   g) Cardiopulmonary bypass

Antidote Administration:

Antidotes work in any one of a number of ways. Common modes of action are as follows :

1. **Inert complex formation:** Some antidotes interact with the poison to form an inert complex which is then excreted from the body e.g., chelating agents for heavy metals, Prussian Blue for thallium, specific antibody fragments for digoxin, dicobalt edetate for cyanide, etc.

2. **Accelerated detoxification:** Some antidotes accelerate the detoxification of a poison e.g., thiosulfate accelerates the conversion of cyanide to non-toxic thiocyanate, acetylcysteine acts as a glutathione substitute which combines with hepato-toxic paracetamol metabolites and detoxifies them.

3. **Reduced toxic conversion:** The best example of this mode of action is provided by ethanol which inhibits the metabolism of methanol to toxic metabolites by competing for the same enzyme (alcohol dehydrogenase).

4. **Receptor site competition:** Some antidotes displace the poison from specific receptor sites, thereby antagonising the effects completely. The best example is provided by naloxone, which antagonizes the effects of opiates at stereo-specific opioid receptor sites.

5. **Receptor site blockade:** This mode of action is best exemplified by atropine which blocks the effects of anticholinesterase agents such as organophosphates at muscarinic receptor sites.

6. **Toxic effect bypass:** An example of this type of antidotal action is provided by the use of 100% oxygen in cyanide poisoning.

Table 1.11 represents a list of genuine antidotes recommended in toxicological practice today.

Nursing Care:

This is especially important in comatose patients, and involves the following measures:

1. Attention to pressure points to prevent the development of decubitus ulcers - hourly turning, a pillow between the legs, use of a ripple mattress if available, etc.
2. In the absence of spontaneous blinking, avoid exposure keratitis by methyl cellulose eye drops, and if necessary, secure the eyelids with adhesive tape.
3. Change bed linen frequently if it gets soaked with urine or stained with faeces.
4. Urinary incontinence can be managed with a sheath urinal for a male, but for a female an indwelling silastic catheter inserted with aseptic precautions is necessary.

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Table 1.11 Specific Antidotes

Antidote	Main Indication	Other Applications
Acetylcysteine	Paracetamol	Amanitin
Amyl nitrite	Cyanide	Hydrogen sulfide
Ascorbic acid	Organic peroxides (Osmium)	----
Atropine	Cholinergic agents	----
Aurintricarboxylic acid (ATA)	Beryllium	----
Benzyl penicillin	Amanitins	----
β aminoproprionitrile	Acids	----
Calcium salts	Oxalates, fluorides	Calcium antagonists
Dantrolene	Malignant hyperthemia	Malignant neuroleptic syndrome
Desferrioxamine	Iron, aluminium	Paraquat
Diazepam	Chloroquine	----
Dicobalt edetate	Cyanide	----
Digoxin specific antibody fragments	Digitalis glycosides	----
Dimercaprol	Arsenic	Copper, gold, mercury
4, Dimethyl aminophenol (4 -DMAP)	Cyanide	Hydrogen sulfide
Ethanol	Methanol, ethylene glycol	----
Flumazenil	Benzodiazepines	----
Glucagon	Beta blockers	----
Glucose	Insulin	----
Guanidine	Botulism	----
Hydroxocobalamine	Cyanide	----
Isoprenaline	Beta blockers	----
Methionine	Paracetamol	----
4, Methylpyrazole	Ethylene glycol, methanol	Disulfiram, Coprin
N-Acetylpenicillamine	Mercury	----
Naloxone	Opiates	----
Neostigmine	Peripheral anticholinergics	----
Oximes	Organophosphates	----

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Oxygen	Cyanide, carbon monoxide, hydrogen sulfide	----
Oxygen (Hyperbaric)	Carbon monoxide	Cyanide, hydrogen sulfide, carbon tetrachloride
Penicillamine	Copper	Gold, lead, mercury
Pentetic acid (DTPA)	Radioactive metals	----
Phentolamine	Alpha adrenergics	----
Physostigmine	Central anticholinergics	----
Phytomenadione (Vitamin K)	Coumarin derivatives	----
Potassium hexacyanoferrate (Prussian Blue)	Thallium	----
Propranolol	Beta adrenergics	----
Protamine sulfate	Heparin	----
Pyridoxine	Isoniazid	Ethylene glycol, gyrometrine, hydrazines
Sodium nitrite	Cyanide	Hydrogen sulfide
Sodium nitroprusside	Ergotism	----
Sodium salicylate	Beryllium	----
Sodium thiosulfate	Cyanide	Bromate, chlorate, iodine
Succimer (DMSA)	Lead, mercury	----
Tocopherol	Carbon monoxide	Oxygen toxicity
Toluidine blue	Methaemoglobinemia	----
Trientine (triethylene tetramine)	Copper	----
Unithiol (DMPS)	Arsenic	Copper, nickel, lead, cadmium, mercury

Modified from **Meredith TJ, Jacobsen D, Haines JA, Berger JC**. Eds. *International Program on Chemical Safety/ Commission of the European Communities Evaluation of Antidotes Series*. Vols 1and 2. Cambridge University Press **1993**.

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5. Inhalation of gastric contents is a frequent problem which can lead to pneumonitis. This can be prevented by positioning the patient semiprone with the head slightly dependent, and intubating if necessary.
6. Adequate bronchial toilet is essential, with regular aspiration of secretions.
7. Passive physiotherapy may be advisable to prevent stiffness and muscle atrophy.
8. Prophylactic antibiotics, if necessary.

Annexure II details the methods of assessing the depth of coma in poisoned patients.

Psychiatric Care

Any patient who has taken an overdose or manifests suicidal ideation should get psychosocial assessment and support as early as possible. The initial evaluation can be performed prior to a total clearing of the patient's sensorium, but a final assessment should not be made unless the patient is completely alert. Recognition that the patient is possibly suicidal, with a precise analysis of the potential for suicide is essential. Carefully analysing the patient's psychologic state (depressed, uncooperative, unresponsive, agitated, anxious, violent, or psychotic), will allow for a realistic appraisal of the psychosocial alternatives with respect to immediate and long term treatment, disposition, and continued follow up, or outpatient care.

MEDICO-LEGAL DUTIES OF A DOCTOR IN SUSPECTED / ACTUAL POISONING[7]

1. If a case of poisoning is indubitably accidental or suicidal in nature, the attending doctor is under no legal obligation to notify the police in case he is working in a private hospital. But if the patient dies, the police has to be informed. Death certificate must not be issued.
2. Doctors working in government hospitals are required to report every case of poisoning regardless of the nature, to the police.
3. All cases of homicidal poisoning (definite or suspected) must be compulsorily reported to the police as per **section 39** of the **Criminal Procedure Code (CrPC)**. Failure to do so will make him culpable under **section 176** of the **Indian Penal Code (IPC)**.
4. If the police require information on any case of poisoning which is either suicidal or homicidal in nature, the attending doctor has to divulge it. There is no scope for professional secrecy in such matters (**175 CrPC**). If information is with-held or wrong information is provided, the doctor becomes culpable under section **202** and **193 IPC** respectively.
5. Every effort must be made by the attending doctor to collect and preserve evidence suggestive of poisoning. Deliberate omission to do so can attract punishment under section **201 IPC**.
6. Collect vomitus, faeces, stomach washings, contaminated food, incriminating substance, etc., and despatch the same for chemical analysis to the nearest Forensic Science Laboratory.
7. If a poisoned patient is conscious but on the verge of death, record a dying declaration relating to the circumstances. It is preferable to call a magistrate for this purpose, but if death appears imminent, or if there is likelihood of delay in the arrival of the magistrate, the attending doctor must himself record the declaration as per **section 32, clause 1**, of the **Indian Evidence Act (IEA)**. Even when a declaration is taken down by a magistrate, the presence of a doctor is desirable to certify that the dying victim was in possession of his senses, and there was no clouding of judgement or coherence which is sometimes encountered in the final moments before death.

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8. If a patient dies before the exact diagnosis could be made out, or he was brought dead to the hospital, the duty doctor must notify the police who will in all probability order an autopsy to be done. Death certificate must not be issued.

9. Detailed written records should be made with respect to every case of poisoning and kept in safe custody.

10. If a doctor comes across a case of food poisoning from a public eatery (canteen, café, restaurant, etc), he must notify the public health authority concerned.

INDIAN STATUTES ON DRUGS / POISONS[8]

Several legal acts have been passed regulating and controlling the manufacture, sale, distribution, and possession of drugs and poisons. The principal acts include the following:

1. **The Poisons Act (1919):** This Act was amended in 1958 and repealed in 1960. It deals with the import of poisonous substances into India, license issuance for possession of certain specified poisons, and restrictions in the sale of such substances (mostly chemicals) as poisons, over which control is to be exercised.

2. **Drugs and Cosmetics Act (1940):** This Act was amended in 1964. It deals with the import, manufacture, distribution, and sale of all kinds of drugs. As per the Act, every patented or proprietary medicinal preparation should display on the label of the container, either the exact formula or a list of the ingredients.

3. **The Drugs and Cosmetics Rules (1945):** This is an offshoot of the **Drugs and Cosmetics Act of 1940**, and is concerned mainly with the standard and quality of drugs, apart from exercising control over the manufacture, sale, and distribution, of drugs and cosmetics. All types of drugs used in therapeutics have been included : allopathic, homeopathic, ayurvedic, unani, and siddha. All drugs and cosmetics are required to be labelled and packed appropriately.

The Drugs and Cosmetics Rules have classified drugs into various Schedules as follows:

- *Schedule C* – Biological products, sera, vaccines, etc.
- *Schedule E* – List of poisonous substances under ayurvedic, siddha, and unani systems.
- *Schedule G* – Hormonal preparations, antihistamines, anti-cancer drugs.
- *Schedule H* – Barbiturates, amphetamines, reserpine, ergot, and some sulphonamides.
- *Schedule J* – Drugs claimed to prevent or cure ailments such as appendicitis, blindness, cancer, cataract, epilepsy, hydrocoele, etc., which must not be advertised or imported.
- *Schedule L* – List of prescription drugs (which includes drugs from Schedule H also)

4. **The Pharmacy Act (1948):** The objective of this Act is to allow only registered pharmacists to compound, prepare, mix, or dispense any medicine on the prescription of a registered medical practitioner. Under this Act, the **Pharmacy Council of India** has been constituted which regulates the study of pharmacy throughout the country. Individual states have **State Pharmacy Councils** for registration of pharmacists.

5. **The Drugs Control Act (1950):** This Act regulates the supply and distribution of drugs, and also guides the manufacturer or dealer in fixing the maximum price for every drug.

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6. **The Drugs and Magic Remedies (Objectionable Advertisement) Act (1954):** The objective of this Act is to ensure that ethical standards are maintained when drugs are advertised by the manufacturers. Advertisements which offend decency or morality can be banned under this Act. Also, those which claim magical powers for certain drugs, e.g., enhancement of potency, cure for incurable diseases, etc. Magical remedies include the use of talismans or charms such as "mantras", "kavachas", etc.

7. **The Narcotic Drugs and Psychotropic Substances Act (1985):** The Narcotic Drugs and Psychotropic Substances Act (NDPS Act) was enacted in India and subsequently amended in 1988, to implement the provisions of the Convention on Psychotropic Substances (1971), and the Convention Against Illicit Traffic in Narcotic Drugs and Psychotropic Substances (1988), both held in Vienna. This Act repeals and renders obsolete three previous Acts – the **Opium Act (1857)**, the **Opium Act (1878)**, and the **Dangerous Drugs Act (1930)**.

   The term "narcotic" in the legal sense is quite different from that used in the medical context which denotes a sleep-inducing agent. Legally, a narcotic drug could be an opiate (a true narcotic), cannabis (a non-narcotic), or cocaine (the very antithesis of a narcotic, since it is a stimulant). The term "psychotropic substance" is with reference to mind-altering drugs such as LSD, phencyclidine, amphetamines, barbiturates, methaqualone, benzodiazepines, mescaline, psilocybine, and designer drugs (MDMA, DMT, etc).

   The NDPS Act imposes complete prohibition on the cultivation of coca, poppy, and cannabis plants, and the manufacture, sale , purchase, use, or transport of any narcotic drug or psychotropic substance except for medical or scientific purposes.

   The minimum punishment for any offence committed under the Act is 10 years rigorous imprisonment and fine of Rs.1 lakh, while the maximum punishment is 20 years rigorous imprisonment and fine of Rs.2 lakhs. There is also sufficient scope under the NDPS Act for enhanced punishment for repeat offences especially after previous convictions, which includes the imposition of even the death penalty [9]. To constitute an offence the first time around, the minimum quantity seized should be equal to or over 250 mg for heroin, 5 gm for hashish or charas, 5 gm for opium, 125 mg for cocaine, and 500 gm for ganja.

   While the NDPS Act generally prohibits the cultivation of poppy, cannabis, and coca plants, it does not impose a total ban. Restricted cultivation of these plants is allowed under strict control for scientific or medical use. Prior sanction in the form of license is necessary from the Central Government for this purpose. For instance, poppy can be cultivated only in certain specified tracts in the states of Rajasthan, Uttar Pradesh, and Madhya Pradesh during a specified period, the *opium year* commencing on the first day of October every year, and ending on the 30th day of September the following year.

TOXICOLOGY AND THE CRIMINAL LAW

The following sections of the **Indian Penal Code (IPC)** deal directly with offences involving poisons:

- **284:** This section deals with negligent conduct in relation to poisons, and states, "Whoever does with any poisonous substance any act in any manner so rash or negligent as to

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endanger human life or to be likely to cause hurt or injury to any person, or knowingly or negligently omits to take such order with any poisonous substance in his possession as is sufficient to guard against any probable danger to human life from such poisonous substance, shall be punished with imprisonment (up to 6 months), or fine (up to Rs.1000), or both."

The gist of this offence is culpable negligence with respect to poisonous substances. The fact that a person has the custody of any dangerous substance suffices itself to impose upon him the duty of being careful.

- **328:** Like section 284, this section deals specifically with poisons. It states, "Whoever administers to any person any poison, or any stupefying, intoxicating, or unwholesome drug with intent to cause hurt to such person, shall be punished with imprisonment (up to 10 years), and shall also be liable to fine."

Apart from these sections, there are other non-specific sections of the IPC which can also be applied in cases of poisoning, for e.g., **299, 300, 302, 320, 324,** and **326.**

POSTMORTEM EXAMINATION IN A CASE OF POISONING

Poisoning cases being invariably medico-legal in nature, if the patient dies an inquest will have to be done, followed by postmortem examination by a forensic pathologist. This is for the purpose of ascertaining the circumstances in which poisoning occurred, and to establish the exact cause and manner of death.

The general procedure of examination is the same as for any medico-legal autopsy, with particular attention being paid to those aspects which can afford a clue to the detection of and identification of the poison involved. The following is a summary of the important points to be noted:

External Examination

1. Stains or marks of vomit, faecal matter, etc., on the clothing, or on the body.
2. There may be evidence of corrosion in the form of discolouration and sloughing especially around the mouth, in caustic ingestion (acid/alkali).
3. Presence of jaundice suggests a hepatotoxic poison, or one which causes haemolytic anaemia.
4. Odour: Several poisons have characteristic odour which may be perceptible in the vicinity of the mouth. (**Table 1.4**, page no. 280)
5. Colour of postmortem lividity: Certain poisons impart characteristic colouration, for example:

▪ Carbon monoxide	*Cherry pink*
▪ Cyanide	*Brick red*
▪ Hydrogen sulfide	*Greenish blue*
▪ Phosphorus	*Brown*
▪ Nitrobenzene, aniline, potassium chloride	*Brownish red*

6. Putrefactive changes: Some poisons are said to retard the rate of decomposition of a dead body, e.g., arsenic, organophosphates.
7. Injection marks: Especially likely in a victim who had been a drug addict in life.

Internal Examination

1. Odour: It is preferable to open the cranial cavity first, since poisons impart a faint odour to the brain which may be difficult to perceive in the presence of overpowering odours from the thorax or abdomen if they have been opened earlier. Examples of such poisons include alcohol, chloroform, cresol, cyanide, and phenol.

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2. Evidence of inflammation: Ingested poisons may cause softening, reddening, corrosion, or even perforation of the gastrointestinal tract. Sometimes the poisonous substance in the form of tablets, powder, plant parts, or fluid may still be present. There may be associated odour.

3. The state of the other organ systems: Liver and kidneys are invariably congested. In some cases, there is evidence (gross or microscopic) of degenerative changes or even necrosis. Brain may be congested or oedematous, particularly in the case of neurotoxic poisons. Petechiae in the white matter are often seen with asphyxiant poisons, which also produce pulmonary oedema with consequent froth in the airways. The heart may demonstrate petechiae or degenerative changes in the case of cardiotoxic poisons. Subendocardial haemor-

Table 1.12 Viscera and Body Fluids to be Preserved in Suspected Poisoning

1. Routine Viscera & Body Fluids - (to be preserved routinely for analysis, in every case of poisoning)

Specimen	Quantity
1. Stomach	Entire
2. Stomach contents	Entire (preferably)
3. Small intestine (jejunum)	30 cm (entire length in infants)
4. Small intestinal contents	Up to 100 gm
5. Liver (portion containing gall bladder)	500 gm (entire liver in infants)
6. Kidney	One half of each kidney (both kidneys in infants)
7. Urine	30 – 50 ml
8. Blood	10 – 20 ml

2. Special Viscera & Body Fluids - (all or any of these should be preserved in addition to routine viscera, in certain specific cases of poisoning)

Specimen	Quantity	Poison
1. Cerebrospinal fluid	As much as can be withdrawn	Alcohol
2. Brain	Half (one hemisphere)	Alcohol, anaesthetics, barbiturates, carbon monoxide, cyanide, opiates, strychnine
3. Spinal cord	Entire length	Gelsemine, strychnine
4. Lung	One	Inhaled poisons
5. Skin (with underlying tissue)	Affected portion	Corrosives, injected poisons
6. Long bone (preferably femur)	10 cm length	Heavy metals
7. Scalp hair (plucked)	15 to 20 strands	Heavy metals

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rhages are said to be characteristic (though not pathognomonic) of acute arsenic poisoning.

CHEMICAL ANALYSIS[10]

In every case of death due to poisoning, an attempt must be made to demonstrate the presence of poison by standardised analytical methods. For this purpose, the pathologist conducting the autopsy must collect certain of the viscera and body fluids, and despatch them through the police to the nearest forensic science laboratory. While submitting the samples for analysis it must be ensured that the correct quantity has been preserved in appropriate preservative in suitable, sealed containers. The materials to be collected are mentioned in **Table 1.12**, while the recommended preservatives are mentioned in **Table 1.13**

Table 1.13 Preservatives for Viscera and Body Fluids

Specimen	Preservative
1. Viscera	Rectified spirit* or saturated saline
2. Blood	For every 10 ml, use 30 mg potassium oxalate and 100 mg sodium fluoride**
3. Urine	100 mg sodium fluoride for every 10 ml

** Not to be used in poisoning due to alcohol, phosphorus, acetic acid, phenol, and paraldehyde.*
*** For carbon monoxide and other gaseous/volatile poisons, use glass or plastic tube, and ensure that there is very little head space after filling.*

HISTOPATHOLOGICAL EXAMINATION

Since poisons can cause degenerative changes in target organs, histopathological evidence of such damage can be a valuable corroborative adjunct. Microscopic examination of tissues may also sometimes help to substantiate a suspicion of long standing abuse which could have contributed to the cause of death. Tissues submitted for histopathology must always be preserved in formalin.

ANALYTICAL METHODS IN TOXICOLOGY

QUALITATIVE ASSAYS

I. **Bedside tests**

A. **Colour Tests**

1. **Trinder's Test**: Add 100 ml of Trinder's reagent (40 gm mercuric chloride in 850 ml water and 120 ml aqueous hydrochloric acid mixed with 40 gm hydrated ferric nitrate diluted to 1 litre with warm water), to 2 ml urine, and mix for 5 seconds.

   A violet or purple colour indicates the presence of salicylates (salicylic acid, salicylamide, and methyl salicylic acid). If the specimen merely darkens, the result is considered negative.[6]

   If only stomach contents or scene residues are available, hydrolyse by heating with 0.5 mol/L hydrochloric acid in a boiling water bath for 2 minutes, and neutralise with 0.5 mol/L sodium hydroxide before performing the test.

2. **Ferric Chloride Test**: Add 1 ml of 5 % ferric chloride solution to 2 ml urine. A persistent purple colour indicates the presence of phenol, phenothiazines, phenylbutazone, oxyphenbutazone, or salicylates.

3. **Lee Jones Test:** Add a few crystals of ferrous sulfate and 4 to 5 drops of 2 %

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sodium hydroxide to 5 ml gastric fluid. Boil and cool. Add 8 to 10 drops of 10 % hydrochloric acid.

A greenish blue colour indicates cyanide, while purple colour indicates salicylates in the sample.

4. **Qualitative Desferrioxamine Colour Test (QDCT):** 2 ml of gastric fluid and 2 drops of 30 % hydrogen peroxide are placed in two plastic tubes. 5ml of desferrioxamine solution (500 mg in 4 ml of distilled water) is placed in one tube, and the resulting colour change is compared with the other tube (control).

   If an orange or red colour develops, it indicates the presence of toxic levels of iron. The test must be done within 2 hours of poisoning or else it is unreliable.[7]

   A variation of this test is the **Desferrioxamine Challenge Test** (DCT), in which 25 to 50 mg / kg of desferrioxamine is administered to the patient by intramuscular injection. Iron poisoning is indicated if the urine which is voided subsequently is pinkish in colour (*vin rose ' urine*).

5. **Meixner Test**: This test can be done on either stool or gastric sample. Dilute the sample with methanol, centrifuge and filter it. Add a drop or two to a piece of newspaper. Encircle the spot with a pencil and dry it. Add a few drops of concentrated hydrochloric acid to the spot

   If a blue colour forms within a few minutes, it is indicative of the presence of amatoxin (present in most toxic mushrooms).

B. **Other Tests**

1. **Isonitrile Test:** Mix a small amount of gastric contents with 10 ml water and add 1 ml purified aniline, followed by 2 ml 20% sodium hydroxide. Heat gently for a few minutes.

   A foul odour (skunk odour) will be perceived if one of the following poisons is present: carbon tetrachloride, chloral hydrate, chloroform, methyl bromide, or any other chlorinated hydrocarbon.

2. **Tensilon Test (Edrophonium Challenge Test)**: When 10 mg edrophonium is given intravenously in a case of sudden paralysis, there will be dramatic recovery if it is due to myasthenia gravis, while a case of poisoning (e.g., botulism) will not show any improvement.

3. **Melzer's Test:** This is a test done to confirm whether a given mushroom is toxic (especially Amanita phalloides). The spores obtained from the mushroom are stained with 1 drop of Melzer's reagent (mixture of 20 ml water, 1.5 gm potassium iodide, 0.5 gm iodine, and 20 gm chloral hydrate), and viewed under a microscope. Spores of A.Phalloides and a few other deadly mushrooms will show a bluish black colour (amyloid reaction). However, a negative reaction does not mean that the mushroom is non-toxic[8].

II. **Thin layer chromatography**[11]: This is a qualitative technique which involves the movement by capillary action of a liquid phase (usually an organic solvent) through a thin, uniform layer of stationary phase (usually silica gel)

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held on a rigid support (usually a glass, aluminium, or plastic sheet). Compounds are separated by partition between the mobile and stationary phases.

A very thin layer of silica gel or aluminium oxide is applied to a glass plate 20 x 20 x 0.5 cm, and vertical lines are drawn 1.5 cm apart to allow individual runways for each sample. Purified tissue extracts dissolved in 0.5 ml of methanol are serially spotted with a micropipette and dried in a small circle in the lower centre of a runway 1.5 cm from the bottom. Other samples are similarly spotted in other runways. A horizontal line (stop point) is drawn 10 or 15 cm above these starting points. A TLC tank is filled with suitable developing solvents to a depth of about 1 cm from the bottom. The plate properly spotted is then dipped into the solvent, the lid is firmly closed and the atmosphere is allowed to saturate with vapour. When the solvent front just touches the 10 cm horizontal mark, quickly remove the plate and examine under UV light (at 254 nm and 366 nm) for characteristic fluorescence or absorbance. Calculate the Rf value.

$$\text{Rf is the ratio of } \frac{\text{Solute front}}{\text{Solvent front}} \times 100$$

Approximate quantitation can be done by comparison with standards similarly prepared on the same plate, for intensity of colour and area size. The Rfs suggested in the literature can be used as a guide for identifying various poisons, though it is preferable that each analyst should prepare and establish his own Rf according to his own conditions and technique.

TLC is a simple, inexpensive technique which is widely used. It takes only about 2 hours from beginning to completion. It is also a very versatile method since the order of separation of compounds can be altered simply by changing the nature of the developing agent. However, interpretation of the plates can prove difficult and calls for a trained eye with considerable experience in recognising colours, spot shapes, and metabolite patterns.

QUANTITATIVE TESTS

1. Ultraviolet Spectrophotometry (UVS)
2. Gas Chromatography (GC)
3. High Performance Liquid Chromatography (HPLC)
4. Mass Spectrometry (MS)
5. Radio Immuno Assay (RIA)
6. Enzyme Mediated Immuno Assay Technique (EMIT)
7. Atomic Absorption Spectrophotometry (AAS)
8. Neutron Activation Analysis (NAA)

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CHAPTER - 2

Caustics

The term **caustic*** is often mistakenly presumed to denote an alkali, while actually it has a much broader meaning and refers to any substance which is corrosive and burning in nature. Obviously this would include apart from alkalis, the more important group comprising acids (inorganic and organic). **Table 2.1** lists the common caustic substances encountered in toxicological practice. All caustics are highly injurious locally and produce burns of varying severity and intensity.

ACIDS

Acids are hydrogen containing substances that on dissociation in water produce hydronium ions. They are potent desiccants with the ability to produce coagulation necrosis (or sometimes liquefactive necrosis) of tissues on contact. Ingestion of acid causes more damage to the stomach than the oesophagus because the squamous epithelium of the latter is more resistant to acids, while it is just the opposite in the case of alkali ingestion where the columnar epithelium of the stomach is more resistant. However the current concept is that the minimal oesophageal damage in acid ingestion is more probably because of rapid oesophageal transit and limited penetrating ability, rather than from any special protective properties of the squamous epithelium[1]. Acid burns of the stomach most commonly involve the antrum and pylorus.

A. **Inorganic acids:** Most of the information mentioned in the foregoing section actually pertains to inorganic or mineral acids, while corrosion is not really a prominent feature of organic acids. But on account of better absorption, organic acids display prominent systemic effects (page no. 304).

Sulfuric Acid

Synonym: Oil of Vitriol

Physical Appearance: Sulfuric acid is a heavy, oily, colourless, odourless, nonfuming liquid. It is hygroscopic i.e., it has great affinity for water with which it reacts violently, giving off intense heat.

Uses

1. Industrial chemical (95 to 98 % solution)
2. Storage batteries and accumulators (30 to 35 % solution)
3. Drain cleaner (8 to 10 % solution)

Usual Fatal Dose: 20 to 30 ml of conc. sulfuric acid. Deaths have been reported with ingestion of as little as 3.5 ml.

* *Derived from the Greek word "kaustikos", which means "capable of burning".*

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Toxicokinetics: Systemic absorption of sulfuric acid is negligible.

Mode of Action

Produces coagulation necrosis of tissues on contact.

Clinical Features

1. Burning pain from the mouth to the stomach. Abdominal pain is often severe.
2. Intense thirst. However, attempts at drinking water usually provoke retching.
3. Vomiting: The vomitus is brownish or blackish in colour (due to altered blood), and may contain shreds of the charred wall of the stomach.
4. If there is coincidental damage to the larynx during swallowing or due to regurtitation, there may be dysphonia, dysphagia, and dyspnoea.
5. Tongue is usually swollen, and blackish or brownish in colour. Teeth become chalky white. There may be constant drooling of saliva which is indicative of oesophageal injury.
6. There is often acid spillage while swallowing with consequent corrosion of the skin of the face (especially around the mouth), neck and chest. Burnt skin appears dark brown or black.
7. Features of generalised shock are usually apparent.
8. If perforation of stomach occurs, a severe form of chemical peritonitis can result.
9. If the patient recovers, there are usually long term sequelae such as stricture formation which may lead to pyloric obstruction, antral stenosis, or an hour glass deformity of the stomach. The oesophagus may also be involved resulting in stenosis. There are indications of increased propensity for carcinomas.
10. Contact of the acid with the eyes can cause severe injury including conjunctivitis, periorbital oedema, corneal oedema and ulceration, necrotising keratitis, and iridocyclitis.

Table 2.1 Common Caustics

Acids		Alkalis	Others
Inorganic (Mineral)	Organic		
Boric	Acetic	Ammonia	Hydrogen peroxide
Chromic	Carbolic	Calcium hydroxide	Iodine
Hydrochloric	Citric	Potassium carbonate	Potassium permanganate
Nitric	Formic	Potassium hydroxide.	Quaternary ammonium compounds
Phosphoric	Oxalic	Sodium carbonate	
Sulfuric		Sodium hydroxide	

Diagnosis

1. *Litmus test*: The pH of the saliva can be tested with a litmus paper to determine whether the chemical ingested is an acid or an alkali.
2. Fresh stains in clothing may be tested by adding a few drops of sodium carbonate. Production of effervescence (bubbles) is indicative of an acid stain[2].

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Treatment

1. Respiratory distress due to laryngeal oedema should be treated with 100 % oxygen and cricothyroidotomy.

2. Some authorities recommend administration of water or milk if the patient is seen within 30 minutes of ingestion (120-240 ml in an adult, 60-120 ml in a child). But no attempt must be made at neutralisation with alkalis, since the resulting exothermic reaction can cause more harm than benefit.

3. Remove all contaminated clothes and irrigate exposed skin copiously with saline.

4. Eye injury should be dealt with by retraction of eyelids and prolonged irrigation for at least 15 to 30 minutes with normal saline or lactated Ringer's solution, or tap water if nothing else is available. Anaesthetic agents and lid retractors may be necessary.

5. The following measures are **contraindicated**: oral feeds, induction of vomiting, stomach wash, and use of activated charcoal.

6. Administration of steroids has been shown to delay stricture formation (in animals) when given within 48 hours of acid ingestion, but the practice is generally not recommended because of increased risk of perforation[3]. Administer antibiotics only if infection occurs. Prophylactic use is not advisable unless corticosteroid therapy is being undertaken.

7. Since there is often severe pain, powerful analgesics such as morphine may have to be given.

8. The use of flexible fibreoptic endoscopy is now standard practice in the first 24 to 48 hours of ingestion to assess the extent of oesophageal and gastric damage[4]. If there are circumferential 2nd or 3rd degree burns, an exploratory laparotomy should be performed. If gastric necrosis is present, an oesophagogastrectomy may have to be done.

9. Emergency laparotomy is mandatory if there are signs of perforation and peritonitis.

10. If the patient recovers, there may be long term sequelae such as stenosis and stricture formation. Follow-up is therefore essential to look for signs of obstruction – nausea, anorexia, weight loss. Surgical procedures such as dilatation, colonic bypass, and oesophagogastrostomy may have to be undertaken.

Autopsy Features

1. Corroded areas of skin and mucous membranes appear brownish or blackish. Teeth appear chalky white.
2. Stomach mucosa shows the consistency of *wet blotting paper*, (**Fig 2.1**).
3. There may be inflammation, necrosis, or perforation of the g.i.tract.

Forensic Significance: Accidental poisoning may arise from mistaken identity since sulfuric acid resembles glycerine and castor oil. It is therefore imperative that it is stored in a distinctive bottle, clearly labelled, and kept in a safe place.

Sulfuric acid is a rare choice for either suicide or homicide.

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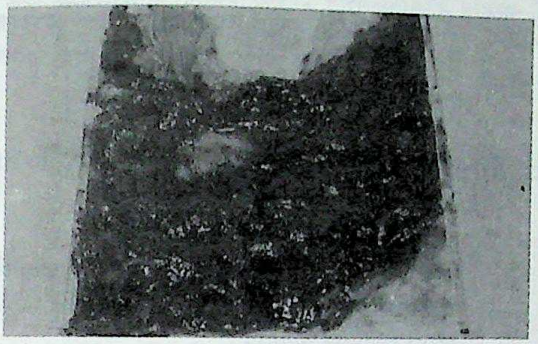

Fig 2.1 Stomach in sulfuric acid poisoning

In addition to routine viscera and body fluids, a portion of corroded skin should be cut out, placed in rectified spirit or absolute alcohol and sent for chemical analysis.

Vitriolage

This term refers to the throwing of an acid on to the face or body of a person in order to disfigure or blind him. The motive is usually revenge or jealousy. This ghastly practice had its origins in the industrial unrest of Glasgow in the 1820s.

Though sulfuric acid is commonly used (hence the term vitriolage which is derived from "oil of vitriol"), other acids are also employed. In fact any corrosive which is easy to hand may be used, including organic acids, alkalis, and irritant plant juices. Going by newspaper reports, vitriolage is a fairly common crime in India, though it is regarded as a serious offence (grievous hurt), and carries stiff punishment.

Nitric Acid

Synonym

Aqua fortis

Physical Appearance: Nitric acid is a colourless or yellowish fuming liquid with an acrid, penetrating odour.

Uses

1. Engraving: Engraver's acid consists of 63 % nitric acid.
2. Electroplating.
3. Manufacture of fertilizers.
4. Metal refinery.

Usual Fatal Dose

20 to 30 ml.

Toxicokinetics

Systemic absorption is negligible.

Mode of Action: Nitric acid is a powerful oxidising agent and reacts with organic matter to produce trinitrophenol, liberating nitrogen monoxide (*xanthoproteic reaction*). Corrosion is less severe when compared to sulfuric acid.

Clinical Features: The general picture is the same as in the case of sulfuric acid, with the following differences :

1. Corroded areas appear yellowish. Stains on clothing and teeth also appear yellowish.
2. More severe eructation and abdominal distension due to gas formation.
3. Perforation of g.i.tract is less common.
4. Inhalation of fumes can produce coughing, rhinorrhoea, lacrimation, and dyspnoea.

Diagnosis

1. Litmus test : (page no. 301)
2. Drop a small piece of copper into the stomach contents and heat it. Pungent, dark brown heavy fumes will emanate if nitric acid is present in sufficient concentration.

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Treatment: Same as for sulfuric acid. Respiratory distress is present more often and requires special attention.

Autopsy Features

1. Corroded areas of skin, teeth, and mucous membranes appear yellowish. Stains on clothing also show yellowish discolouration.
2. Gastrointestinal perforation is less common.

Forensic Significance: Same as for sulfuric acid. Mistaking it for glycerine or castor oil however is rare because it is a fuming liquid.

Hydrochloric Acid

Synonyms

Muriatic acid, Spirit of salts.

Physical Appearance: Hydrochloric acid is a colourless, fuming liquid which may acquire a yellowish tinge on exposure to air.

Uses

1. Bleaching agent (less than 10 % HCL).
2. Dyeing industry.
3. Metal refinery.
4. Flux for soldering.
5. Metal cleaner, drain cleaner.

Usual Fatal Dose

30 to 40 ml.

Diagnosis

1. Litmus test : (page no. 301)
2. If an open bottle of conc. ammonia solution is placed near the stomach contents or vomitus, copious white fumes of ammonium chloride will emanate. Though normal stomach contents contain hydrochloric acid, this is usually too dilute (0.2 to 0.5 %) to vitiate the value of this test.

The rest of the information on HCL is practically the same as for H_2SO_4, except that corroded areas are more likely to be greyish, and symptoms are generally less severe. But respiratory manifestations are more pronounced.

B. **Organic acids:** Organic acids differ from inorganic acids in two major respects[5]:
   1. They are weaker in action locally.
   2. They are better absorbed into the systemic circulation, and therefore have more powerful remote action.

Carbolic Acid

Synonyms

Hydroxybenzene, phenol.

Physical Appearance: Colourless, needle-like crystals which turn pink and liquefy when exposed to air. It is a coal tar derivative (creosote). Commercial phenol is a brownish liquid containing impurities like cresol. Household phenol (often sold as *phenyle*) contains 5% phenol in water. It has a characteristic "hospital odour".

Derivatives: Catechol, cresols, menthol, resorcinol, thymol – *Toxic*

Hexyl resorcinol, naphthol – Less Toxic
Tannic acid – Least toxic

Uses: Carbolic acid was introduced as a disinfectant in the 19th century by Lemaire, and quickly became popular ever since Lord Lister advocated its use in surgery for asepsis. Even today it is popular as a hospital and household disinfectant, even though several safer and more effective alternatives have been developed including cetrimide, chlorhexidine, chloroxylenol, parachlorometaxylenol, terpineol, and xylenol.

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The various uses of carbolic acid are as follows :

1. Antiseptic and disinfectant : especially for sterilising floors, walls, furnishings, glassware, and instruments.
2. Preservative : Phenol is a commonly used preservative in injectable medications, for e.g., glucagon, pethidine, neostigmine, quinidine, and epinephrine.
3. Pharmaceuticals.
4. Manufacture of plastics.
5. Medical uses:
   a. "Face peel" in plastic surgery
   b. Neurolysis for spasticity (by injecting phenol solution into neuromuscular junctions).[6]

Usual Fatal Dose

10 to 30 gm.

25 to 50 ml of household phenol can cause death. Fatalities have been reported even with much less quantities.[7]

Toxicokinetics

Carbolic acid is rapidly absorbed through g.i mucosa, lungs, and even intact skin. Dilution may actually increase absorption and enhance toxicity.[8]

Mode of Action: Carbolic acid is actually a very mild corrosive, but has profound systemic effects after absorption. There is CNS depression, metabolic acidosis, and renal damage.

Clinical Features:

- **Acute Poisoning**

  Local : Skin or mucosal contact results in mild corrosion with hardening and whitish discolouration. However, the white eschar (especially in the skin) drops off in a few days leaving a brown stain. Locally there may be burning pain followed by tingling, numbness, and anaesthesia.

  Systemic:

  GIT - Burning pain, vomiting.

  CNS - Vertigo, convulsions, coma. Pupils are constricted.

  RS - Slow, laboured breathing, progressing to respiratory failure.

  Blood -Haemolysis, methaemoglobinemia.

  Metabolic - Hypothermia, with cold and clammy skin, metabolic acidosis.

  Hepatorenal - Oliguria, with scanty urine which turns greenish on exposure to air because of phenolic metabolites (hydroquinone and pyrocatechol). This is referred to as *Carboluria.* Later there is renal and hepatic failure.

  Deaths have occurred even after skin contact with carbolic acid.[9]

- **Chronic Poisoning** (*Phenol Marasmus*)

  This was common in earlier days among medical personnel when phenol was routinely used as a skin disinfectant. It is characterised by anorexia, weight loss, headache, vertigo, dark urine, and pigmentation of skin and sclerae (*Ochronosis*).[10]

Diagnosis

1. Typical odour in the vicinity of the patient.
2. Urine collected and stored in a transparent container shows a gradual change in colour to brown or green (**Table 2.2** contains a list of poisons and drugs which discolour urine).
3. To 10 ml of urine, add 1 ml of 10 % ferric chloride. A purple or blue colour which persists even on heating indicates phenol poisoning.

Treatment

1. Decontaminate skin by copious washing.
2. Stomach wash can be done preferably with sodium or magnesium sulfate solution.
3. Activated charcoal in the usual manner.
4. Supportive measures.

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Autopsy Features

1. Distinct odour of phenol, especially around the mouth and in the stomach contents.
2. Corroded areas are at first white, but if death has been delayed they turn brownish.
3. Gastric mucosa is greyish white, swollen, and hardened (leathery).
4. Urine is greenish or brownish.

Table 2.2 Poisons / Drugs Which Discolour Urine

Urine Colour	Poison / Drug
Orange	Phenothiazines, rifampicin
Red or Pink	Aniline, ibuprofen, phenothiazines, phenytoin
Purple	Porphyrins
Reddish-brown	Chloroquine, iron, phenacetin, metronidazole
Brownish-black	Homogentisic acid
Yellowish-brown	Furazolidone, nitrofurantoin, primaquine, sulfamethoxazole
Yellow	Fluorescin dye, riboflavin
Yellowish-orange	Warfarin, carrots, vitamin A
Greenish-blue	Chlorophyll Breath Mints (Clorets), methylene blue, thymol,
Brownish-green	Cresol, phenol

Modified from **Raymond JR, Yarger WE** *Algorithm for differential diagnosis of coloured urine.* South Med J **1988**; 81 : 837-841.

Forensic Significance: Most cases of poisoning are accidental in nature arising out of occupational exposure or therapeutic misuse. Formerly, suicidal ingestions were common. Poisoning with phenolic derivatives causes similar but usually less severe manifestations, and must be treated on the same lines as phenol.

Oxalic Acid

Synonym

Ethanedioic acid, Salt of sorrel

Physical Appearance: Colourless, odourless, prismatic crystals with a sour, slightly bitter taste.

Derivatives: Oxalates

Uses

1. Bleaching and cleaning agent
2. Ink remover
3. Rust remover
4. Metal polishing
5. Industry: Ceramic, leather, metallurgic, paper, pharmaceutical, and rubber industries

Usual Fatal Dose

15 to 30 gm of oxalic acid.

Mode of Action: Liquid oxalic acid has moderate corrosive action on skin and mucosa.

Systemic absorption leads to hypocalcemia, since it reacts with calcium in plasma, and insoluble calcium oxalate is precipitated which accumulates in the liver, kidneys, heart, lungs, and blood, and is excreted in the urine (*oxaluria*).

Clinical Features

- *Local*: Whitish or yellowish corrosion (mucosa), or discolouration (skin), with underlying congestion. The corroded mucosa is often referred to as "scalded" in appearance. Production of acid haematin however can turn the mucosa blackish.
- *Systemic*: Vomiting ("coffee grounds"), and diarrhoea, followed by signs and symptoms of hypocalcemia (tetany), characterised by tonic muscle spasms, cramping, and

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accoucher's hand.* There is often a positive **Trousseau's**** and **Chvostek's***** **sign.** Pupils are usually dilated. Later there may be metabolic acidosis, ventricular fibrillation, and renal failure.

Diagnosis

1. Demonstration of urinary oxalate crystals which may occur either as monohydrates (prism or needle-like), or dihydrates (tent or envelope shaped).
2. Estimation of serum calcium and oxalate levels.

Treatment

1. Stomach wash with calcium gluconate or lactate solution.
2. Calcium gluconate i.v (10 ml, 10% soln)
3. Dialysis or exchange transfusion for renal failure.
4. Affected skin or eye should be washed copiously with water.

Autopsy Features

1. "Scalded" mucosa of g.i tract (especially stomach). Sometimes there is a brown or black colour due to acid haematin. Crystals of calcium oxalate may be demonstrated in scrapings of the mucosa, (examine with polarising microscope).
2. Whitish or yellowish discolouration of corroded areas.

Forensic Significance: Most cases of oxalic acid or oxalate poisoning result from accidental causes, for e.g., mistaking the substance for Epsom salt, sodium bicarbonate, etc. Accidental poisoning may also result from excessive ingestion of certain vegetables rich in oxalates (rhubarb leaves, sorrel, etc). Chronic consumption can lead to renal calculi with consequent renal colic.

Occasional cases of suicide and homicide with oxalic acid have been reported.

ALKALIES

Alkalies commonly encountered in poisoning include ammonia (usually in the form of ammonium hydroxide), carbonates of sodium and potassium, and hydroxides of sodium, potassium, and calcium. Sodium hypochlorite is also increasingly being implicated.

Physical Appearance

Most of these occur as white powders or colourless solutions. Ammonia gas is colourless with a pungent, choking odour.

Uses

- Ammonia gas – Smelling salts.
- Ammonium hydroxide (32.5% ammonia) – Paint, oil, and dirt remover, refrigerant.
- Sodium hydroxide (Caustic soda) – Drain cleaner, oven cleaner.
- Potassium hydroxide (Caustic potash) – Drain cleaner, hearing aid batteries.
- Sodium carbonate (Washing soda) – Household cleaning agent, detergent.
- Potassium carbonate – Household cleaning agent.
- Sodium hypochlorite – Household bleach.

Usual Fatal Dose

10 to 15 gm for most alkalies.
15 to 20 ml for ammonia.

** Due to carpopedal spasm.*
*** Trousseau's sign : Spasm resulting from pressure applied to nerves and vessels of upper arm.*
**** Chvostek's sign : Spasm of facial muscles following a tap on the facial nerve area.*

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Mode of Action

Locally, alkalies produce liquefaction necrosis which results in extensive penetrating damage because of saponification of fats and solubilisation of proteins. Production of ulcers is common which may persist for several weeks. Oesophagus is more severely affected than the stomach in contrast to acids (page no. 300).

Clinical Features

1. Corrosion of the g.i mucosa with greyish pseudomembrane formation. Oesophagus is often severely affected resulting in dysphagia, vomiting, drooling, and haematemesis.
2. Abdominal pain, diarrhoea, tenesmus.
3. Skin involvement results in greyish, soapy, necrotic areas without charring.
4. Eye involvement can produce serious complications, and constitutes an ophthalmologic emergency.
5. Ammonia ingestion causes manifestations which are essentially similar to those seen with other alkalies, but respiratory symptoms are commonly super-added due to inhalation of fumes while swallowing.

Treatment

1. Respiratory distress (especially in the case of ammonia) may require endotracheal intubation, cricothyroidotomy, or tracheostomy, depending on severity. Oxygen must be administered as necessary.

2. Diluents such as milk or water may be given as a first aid measure for alkali ingestion (no more than one or two glasses for an adult). Neutralisation with acidic solutions is contraindicated since the resultant exothermic reaction can enhance the risk of perforation. However, if action can be taken within 5 minutes of ingestion, neutralisation may help.[11]

3. The following are absolutely **contraindicated**: emesis, gastric lavage, catharsis, and activated charcoal.

4. Withhold all oral feeds initially.

5. Assess fluid and electrolyte balance.

6. Watch for development of complications and attend to them accordingly.

7. Some investigators recommend early surgical intervention and use of an intraluminal stent in patients with 2nd or 3rd degree oesophageal burns because perforation which requires surgical repair may otherwise be missed.[12]

8. If circumferential 2nd degree or 3rd degree burns of the oesophagus are seen, exploratory laparotomy should be undertaken followed by gastric resection and oesophagectomy, in case gastric necrosis is evident at laparoscopy.

9. In the past, administration of corticosteroids was recommended to prevent stricture formation which was based on animal studies.[13] But today most investigators appear to be of the opinion that high dose corticosteroid therapy in caustic ingestion predisposes the patient to infection and perforation as well as masks symptoms of developing peritonitis or mediastinitis, and hence should be avoided. However, steroids may be effective in patients with dyspnoea, stridor, hoarseness, and other evidence of respiratory compromise, and may decrease laryngotracheal oedema thereby lessening respiratory dysfunction.[14]

10. Use of prophylactic antibiotics is not recommended today. Most investigators feel that antibiotics should be administered only if there are signs of perforation or secondary infection.[15]

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11. An oesophagogram should be done at 3 weeks to evaluate the formation of strictures. Patients should be instructed to seek medical attention immediately whenever they develop dysphagia.

12. Alkali injuries to the eye and skin should be irrigated copiously with water or saline for at least 20 to 30 minutes. Ophthalmologic consultation is mandatory. Topical antibiotics and steroids may help.

13. Patients who have suffered from stricture formation require long-term endoscopic follow-up for the presence of neoplastic changes of the oesophagus which may occur with a delay of several years or decades.

Autopsy Features

1. Characteristic odour (in the case of ammonia).
2. Brownish or greyish staining of skin.
3. Inflammatory oedema with corrosion and sliminess of the tissues of the oesophagus and stomach. The mucosa may be brownish owing to formation of alkaline haematin.
4. Congestion of respiratory tract and pulmonary oedema (especially in the case of ammonia).

Forensic Significance

1. Accidental poisoning occurs usually by mistaking an alkali solution for water, lemonade, beer, etc., because of careless storage of these chemicals in inadequately labeled, ordinary looking bottles or jars.
2. Industrial accidents involving these substances are reported from time to time.
3. Suicidal cases are occasionally encountered. Homicides are quite rare.
4. Ammonia may sometimes be sprayed or thrown on a victim to facilitate robbery.

Iodine

Physical Appearance: Blue-black, glittering crystals which constantly give off violet coloured vapour with a peculiar odour.

Uses

1. Antiseptic preparations:
   Lugol's iodine, Tincture of iodine, Povidone-iodine.
2. As a component of some expectorants (e.g., potassium iodide, iodinated glycerol), anti-asthmatics (e.g., iophylline), anti-arrhythmics (e.g., amiodarone), anti-amoebics (e.g., iodoquinol), antifungals (e.g., vioform), anti-thyroid drugs (e.g., potassium iodide), and radiographic contrast agents (e.g., diatrizoate sodium, iopanoic acid, ipodate sodium, etc).

Usual Fatal Dose

2 to 5 gm of free iodine, or 1 to 2 ounces of strong iodine tincture.

Mode of Action

Locally, strong iodine solution can be an intense irritant. Systemic toxicity is due to combination of free iodine with serum sodium bicarbonate, leading to metabolic acidosis.[16]

Clinical Features

Initial manifestations of iodine poisoning include rhinorrhoea, conjunctivitis, and cough (especially if fumes have been inhaled). There is burning pain extending from the mouth to the abdomen, salivation, metallic taste, vomiting, and diarrhoea. Vomitus and stools may appear yellowish (or sometimes bluish) in colour. Skin and mucous membranes are stained yellowish brown.

Iodides and iodophores (e.g., povidone-iodine), are much less toxic and usually require only supportive treament. But chronic poisoning can result from long term therapeutic intake of iodide salts leading to **iodism** which is characterised by metallic taste, anorexia, insomnia, lymphadenopathy, parotid swelling (**iodide mumps**), stomatitis, conjunctivitis, rhinorrhoea, and skin manifestations (erythema, urticaria, acne, etc., together referred to as *ioderma*).

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Diagnosis

Iodine

1. Yellowish stains of clothing, skin, and mucosa.
2. Characteristic odour.
3. Vomitus or stomach contents when heated in a beaker will cause the iodine to sublime on a cold surface (e.g., a watch glass placed on top of the beaker with some ice).

Iodides: To 10 ml of vomitus or gastric aspirate, add 1 ml of 10 % silver nitrate solution and 1 ml nitric acid. Formation of a yellow precipitate which is insoluble in ammonia is characteristic of iodides. Whitish precipitate (soluble in ammonia) is characteristic of chlorides, while bromides produce brown precipitate which is insoluble in ammonia.

Treatment

1. Decontamination:
   - Skin: Wash thoroughly with soap and water, or 20 % alcohol.
   - Eyes: Irrigate with water for 15 minutes.
   - GIT: If oesophageal injury is not present or suspected, gastric lavage can be attempted with starch solution or 5 % solution of sodium thiosulfate, or even plain milk. Activated charcoal binds iodine, and can be administered.
2. Sodium bicarbonate i.v for metabolic acidosis.
3. Supportive measures: Iodism is treated by ceasing iodide intake while enhancing the intake of sodium chloride which promotes excretion of iodides.

Autopsy Features

1. Brownish or yellowish stains of skin and mucosa.
2. Characteristic odour (rarely perceptible).
3. Congestion of viscera, especially the kidneys.

Forensic Significance

Most cases result from accidental therapeutic exposure where iodine or iodides had been administered in excess. Deaths have been reported even with excessive local applications of povidone-iodine.[17] The commonest abnormalities reported with such iodophore applications (repeatedly) are acid-base disturbances and metabolic acidosis.

Chronic poisoning can occur from prolonged therapy with iodides, or may be a result of occupational exposure.

Iodinated radiologic contrast agents are well known to produce anaphylactic reactions.

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CHAPTER - 3

Inorganic Elements

There are innumerable substances which can be included under the term inorganic non-metallic elements. But this chapter concerns itself only with some of the relatively important (or common) examples encountered in toxicological practice such as **phosphorus** and the **halogens**.

Phosphorus

Physical Appearance

There are two main varieties:

1. **Yellow (or White) Phosphorus**: This is a yellowish, waxy, crystalline solid with a garlicky odour. On exposure to air, it oxidises into whitish fumes of phosphorus pentoxide. Hence it is generally stored under water. Yellow phosphorus is highly combustible and ignites into flame at 34°C. It is luminescent and glows in the dark (phosphorescence).

2. **Red Phosphorus**: This is a reddish or brownish, amorphous, odourless substance. It is insoluble and harmless, since it is not absorbed from the g.i tract.

Derivatives and related compounds of phosphorus include phosphoric acid, phosphine, aluminium phosphide, and zinc phosphide.

Uses

1. **Matches:** Yellow phosphorus was extensively used in the manufacture of friction matches during the 19th century. However, because of its propensity to produce chronic poisoning in workers of the match industry, most countries agreed at an international convention in Berne, Switzerland in 1906 to prohibit the manufacture and import of yellow phosphorus for the making of matches. Hence, these so-called "lucifer matches" gradually (and fortunately) faded out. Today's "safety match" contains only potassium chlorate and antimony sulfide. It has to be struck against a prepared surface to ignite it, which is provided by the sides of the match box being coated with powdered glass and red phosphorus. This is in contrast to the "strike anywhere" match which does not need a prepared surface to ignite it. Its composition is 6 % phosphorus sesquisulfide (non-toxic), 24% potassium chlorate, and small quantities of zinc oxide, red ochre (a pigment), powdered glass, glue, and water.

2. **Fireworks:** Although the use of yellow phosphorus in fireworks is prohibited in western countries, it is still an important ingredient in several types of fireworks manufactured in India.

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3. **Military Uses:** Yellow phosphorus is an ingredient of tracer bullets, incendiary bombs, smoke screens, and air-sea rescue flares.

4. **Insecticide and Rodenticide:** There are several pastes and powders available in India which contain phosphorus (or zinc phosphide) used for killing cockroaches and rats (e.g., *Ratol*). Such pastes are usually mixed with molasses or butter and spread on bread as bait. Obviously, unintentional ingestion by children is quite possible leading to severe poisoning.

5. **Fertilizer.**

Usual Fatal Dose

60 mg (roughly 1 mg/kg body weight).

Mode of Action

Yellow phosphorus, a protoplasmic poison is a potent hepatotoxin (**Table3.1**). In large doses it can cause shock and cardiovascular collapse since it is also toxic to the heart. Locally, it produces severe irritation of skin and mucosa. Rate of absorption is greatly enhanced if phosphorus is administered in an oily vehicle.

Clinical Features

A. **Fulminant Poisoning:** This results from ingestion of a massive dose i.e., more than 1 to 2 gm. The dominant clinical picture is one of peripheral vascular collapse. Death usually occurs in 12 to 24 hours, and signs of hepatic or renal damage are not seen.

B. **Acute Poisoning:** Today, most cases of phosphorus poisoning fall in this category. The clinical manifestations characteristically occur in three stages.

- First Stage (upto 3 days): Local effects include severe burning pain, vomiting, diarrhoea, and abdominal pain. Breath smells of garlic. Vomitus and stools may be luminous in the dark. There may be haematemesis. Faint fumes may emanate from the stools (*Smoky stool syndrome*).

- Second Stage (upto several days after the first stage subsides): This is an essentially symptom-free (treacherous) period, and the patient may feel well enough to be discharged from the hospital.

Table 3.1 Hepatotoxic Agents

Acute Hepatocellular Injury	Cholestasis	Steatosis	Chronic Active Hepatitis
Arsenic Carbon tetrachloride Ethyl alcohol Halothane Iron Isoniazid Methyldopa Paracetamol Phenytoin Phosphorus (Yellow) Tetracycline	Chlorpromazine Erythromycin Nitrofurantoin Rifampicin	Ethyl alcohol Nucleoside analogues (Zidovudine) Sodium valproate	Isoniazid Methyl dopa Nitrofurantoin Tetracycline

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- Third Stage: This is due to the systemic effects of phosphorus after it has been absorbed. There is a return of the digestive symptoms with increased severity. In addition, manifestations of liver damage are prominent – tender hepatomegaly, jaundice which may progress to an olive green hue, pruritis, bleeding from multiple sites, and finally hepatic encephalopathy, stupor, and coma. At this stage there is a mousy odour to the breath (foetor hepaticus). Renal damage results in oliguria, haematuria, albuminuria, and acute renal failure.

  Dermal contact with phosphorus results in acutely painful corrosion.

Diagnosis

1. Garlicky odour of breath and vomitus.
2. Fuming or luminous vomitus and stools.
3. Evidence of hepatic and renal failure.

C. **Chronic Poisoning**: This usually results from long term occupational exposure to the fumes of phosphorus pentoxide and results in the condition called *Phossy Jaw* or *Glass Jaw*. Main features include toothache (usually originating in a carious tooth) which because of its recurrence would be eventually extracted leading to exposure of bone followed by necrosis, sequestration, and osteomyelitis of jaw (invariably the lower jaw).

Treatment

A. **Acute Poisoning:**
   1. Gastric lavage with potassium permanganate (1 : 5000), which oxidises phosphorus into relatively less toxic phosphoric acid and phosphates. Some authorities recommend administration of copper sulfate solution (250 gm in a glass of water), which converts phosphorus to non-toxic copper phosphide.[1] Alternatively, a 0.2% solution of copper sulfate may be used for stomach wash.
   2. Do not administer milk or any oily / fatty foods, since this will enhance the absorption of phosphorus.
   3. Vitamin K by i.v drip (65 mg) slowly, to combat hypoprothrombinaemia.
   4. Intravenous fluids.
   5. Whole blood / fresh frozen plasma to correct coagulation defects.
   6. Steroids and ionotropic support for shock.
   7. Treat dermal burns with warm water or 1 % copper sulfate solution followed by application of bland ointments such as white soft paraffin.

B. **Chronic Poisoning:**
   1. Removal of patient from source of exposure.
   2. Dental treatment and follow-up.

Autopsy Features

1. Garlicky odour in the vicinity of the mouth and in the gastric contents.
2. Jaundice.
3. Bleeding points in the skin (subcutaneous haemorrhages).
4. Luminous gastric contents. The contents will fluoresce under u.v light.
5. Congestion and inflammation of affected skin and mucosa.
6. Enlarged fatty liver. Later there is evidence of acute yellow atrophy.

Viscera for chemical analysis must be preserved in saturated saline and not rectified spirit, otherwise luminosity especially of the stomach contents will be lost.

Forensic Significance

1. Accidental Poisoning: This used to be common in the past because of unrestricted use of phosphorus in matches and fireworks. Today,

[1] Alternatively, a 0.2%

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most cases of accidental poisoning result from inadvertent ingestion of cockroach or rat poison by children, or because of contamination of food by these substances.

2. Suicidal Poisoning: This was also previously quite common, especially in western countries. A popular method appears to have been to soak several "lucifer" match heads in water or brandy, mix with sugar, and consume the resultant potion.[2]

   Today, rat pastes containing phosphorus are occasionally implicated in suicidal ingestions.

3. Homicidal Poisoning: Formerly, phosphorus was quite frequently employed for committing murder. Several accounts are mentioned in the literature where poisoning was accomplished by mixing phosphorus in soup, jam, or rum, and administered to unsuspecting victims.

Aluminium Phosphide

Physical Appearance

Aluminium phosphide is marketed in India under various trade names (*Alphos, Celphos, Chemfume, , Fumigran, Phosphotek, , Phostoxin, Quickphos*, etc).

It is generally available as grayish green tablets of 3 gm each, mixed with urea and ammonium carbonate. These tablets are sold in sealed, airtight containers of tens and twenties. Each tablet liberates 1 gm of phosphine.[3]

Uses

Grain preservative: Aluminium phosphide is said to be the most ideal grain preservative since it is relatively cheap while being very effective in repelling pests. The required number of tablets are removed from the airtight container and placed among the grain. On exposure to moisture, phosphine is released which percolates among the grain. When fumigated grains are subsequently well aerated, phosphine evaporates rapidly leaving behind virtually no residue.

Usual Fatal Dose

One to three tablets.

Mode of Action

When exposed to air and moisture, aluminium phosphide liberates phosphine which causes multiorgan damage.

$$AlP + 3H_2O \rightarrow Al(OH)_3 + PH_3$$

Clinical Features

Common presenting symptoms include metallic taste, vomiting, garlicky (or fishy) odour of breath, intense thirst, burning epigastric pain, and diarrhoea.[4] In severe cases, there are cardiovascular manifestations such as hypotension, tachy/bradycardia, and ECG abnormalities (sinus tachycardia, ST-T wave changes, bradycardia with heart block, etc). Coma supervenes in later stages. Respiratory distress is invariably present with cyanosis, and cold, clammy skin.

Aluminium phosphide poisoning carries with it a high mortality, with death occuring virtually 90 to 100% of the time in cases of ingestion involving more than 3 tablets.

Diagnosis

1. Garlicky odour in the breath.
2. Liver function tests are often abnormal.
3. ECG changes (mentioned under Clinical Features).
4. **Silver nitrate test**: A piece of filter paper impregnated with 0.1 N silver nitrate solution is used in the form of a mask through which the patient is asked to breathe in and out for 5 to 10 minutes. Blackening of the paper is indica-

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tive of the presence of phosphine in the breath, since silver nitrate is reduced to silver on exposure to it. Similar reaction is also produced by hydrogen sulfide.

Treatment

1. Management of circulatory shock with i.v fluids (4 to 6 L over 6 hours). Dopamine can be given i.v at a dose of 4 to 6 mg/kg/min (maximum 10mg/kg/min).
2. Management of respiratory distress with 100 % humidified oxygen, intubation, and assisted ventilation.
3. Management of metabolic acidosis with sodium bicarbonate (50 mEq/15 min) until the arterial bicarbonate rises above 15 mmol/L.
4. **Magnesium sulfate therapy:**
   Give i.v as a 3 gm bolus, followed by 6 gm infusion over 24 hours for 5 to 7 days. Alternatively, 1 gm can be given i.v to begin with, followed each hour by the same dose for 3 consecutive hours, and then 1 gm every 6 hours for 5 days.

   Magnesium sulfate is said to be beneficial in the management of cardiac arrhythmias. Conventional antiarrhythmic drugs such as digoxin and lidocaine are ineffective.

Though there is often intense thirst, do not administer water since whatever aluminium phosphide is still remaining in the stomach will react with it, releasing phosphine. For the same reason, stomach wash is contraindicated. While activated charcoal can be administered, it should be mixed with sorbitol (and not water), using 240 ml for evey 30 gm.

Autopsy Features [5,6]

There is widespread hypoxic organ damage with congestion and petechiae. Contents of stomach are often haemorrhagic with mucosal shedding, and there is usually an intense garlicky odour. Microscopy reveals necrotic changes in liver and kidneys. Heart shows features of toxic myocarditis with fibrillar necrosis. Lungs may demonstrate evidence of ARDS (adult respiratory distress syndrome) with or without pulmonary oedema.

Forensic Significance

Prior to 1980, aluminium phosphide poisoning was virtually unreported in India. Today it is the leading cause of suicidal (and sometimes accidental) death in northern Indian states such as Punjab, Haryana, Uttar Pradesh, Madhya Pradesh, and Rajasthan.

HALOGENS

Iodine and iodides have been discussed under **Caustics** (page no.309). The other halogens of importance include chlorine, bromine, and fluorine. Only chlorine will be discussed here. All halogens combine with hydrogen to form acids, and with metals to form salts.

Chlorine

Physical Appearance

Chlorine is a greenish yellow gas with a pungent odour.

Uses

1. Bleaching agent for cotton and paper.
2. Water purification.
3. Sewage treatment.
4. Pharmaceutical and cosmetic industry.

Usual Fatal Dose

Concentrations of over 50 to 100 ppm when inhaled can be rapidly fatal.

Mode of Action

Chlorine is an extremely active oxidising agent and causes rapid and extensive destruction of organic tissue.

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Clinical Features

Chlorine is an irritant gas and inhalation provokes rhinorrhoea, lacrimation, coughing, chest pain, and shortness of breath. Major exposure results in laryngeal oedema, stridor, pneumonitis, and pulmonary oedema. In addition, the following features of systemic toxicity are seen : vomiting, vertigo, headache, ventricular ectopic beats, and metabolic acidosis.

Corrosion of teeth may occur if there is prolonged or chronic exposure.

Diagnosis

1. Characteristic odour.
2. Chlorine gas leak into the atmosphere can be detected by opening a bottle of concentrated ammonium hydroxide which will cause the production of heavy, white fumes of ammonium chloride.

Treatment

1. Mild posoning can be managed with bed rest and oxygen administration.
2. Cough can be controlled with codeine and bronchodilators.
3. Nebulised sodium bicarbonate (3.75 % solution) is claimed to be effective in ameliorating respiratory symptoms by neutralising the acid formed when chlorine comes into contact with water in the airways. This can however provoke an exothermic reaction and doubts have been expressed as to its efficacy and safety.[7]
4. The role of corticosteroids in the treatment of pulmonary oedema is also controversial.
5. Severe cases of poisoning will require intermittent positive pressure ventilation.
6. Eye exposure must be treated with copious irrigation of water or saline. Expert ophthalmic consultation is advisable to rule out corneal damage.

Autopsy Features

1. Characteristic odour.
2. Massive pulmonary oedema.
3. Denudation of respiratory epithelium.

Forensic Significance

Most cases of poisoning are accidental arising out of domestic or industrial exposure. Sometimes, exposure occurs at swimming pools where chlorine is often used as a disinfectant.

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CHAPTER - 4

Heavy Metals

Though the general perception is that heavy metal poisoning is uncommon, the actual fact is just the converse. Heavy metal poisoning (acute or chronic) is a major cause of morbidity and mortality all over the world, and India is no exception. Only arsenic, lead, mercury, iron, and copper will be discussed here.

Arsenic

Arsenic is today the commonest source of acute heavy metal poisoning, and is second only to lead in the incidence of chronic toxicity[1].

Physical Appearance

Arsenic is a metalloid i.e., it is an element which resembles a metal in some respects, and is per se not very toxic. However, almost all the salts are toxic to varying degree. **Table 4.1** displays the physical properties and uses of arsenic and its compounds.

Usual Fatal Dose

200 to 300 mg for arsenic trioxide. In general, the pentavalent form of arsenic (arsenate) is less toxic than the trivalent form (arsenite) because it is less water soluble. The most toxic form is arsine gas.

Toxicokinetics and Mode of Action

Arsenic is absorbed through all portals of entry including oral, inhalational, and cutaneous routes. After absorption it is redistributed to the liver, lungs, intestinal wall, and spleen, where it binds to the sulphydryl groups of tissue proteins. Arsenic replaces phosphorus in the bone where it may remain for years. It gets deposited also in hair.

Clinical Features

Mentioned in **Table 4.2** Dermal pigmentation is consistently seen in chronic arsenic poisoning, and may also be encountered in exposure to certain other substances, (**Table 4.3**).

Diagnosis

1. *Urine level*: If the 24 hour excretion of arsenic exceeds 100 mg, it is indicative of toxicity.

2. *Blood level*: This is less reliable than urine level because of short half-life of arsenic in the blood.

3. *Hair level*: Although considered to be an important diagnostic criterion, it may be virtually useless since it cannot discriminate between external deposition and toxic accumulation.[2]

   The best analytical method for detection of arsenic in biological samples is atomic absorption spectroscopy.

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Table 4.1 Inorganic and Organic Arsenicals

Chemical Name	Synonyms	Physical Properties	Uses
Elemental arsenic	Metallic arsenic, grey arsenic	Shiny grey, brittle, metallic looking substance	In alloys
Arsine	Arsenic trihydride, hydrogen arsenide, arseniuretted hydrogen	Colourless gas with garlicky odour	Lead plating, soldering, galvanising, and in electronic components
Trimethyl arsine	Gosio gas	Colourless gas	Present in sewage
Arsenic trioxide	Arsenic oxide, white arsenic, arsenic sesquioxide, arsenious anhydride	White powder (dissolves slowly in water to form arsenious acid)	Manufacture of glass, insecticide, rodenticide. Previously used in medicine for treating fever (e.g., Fowler's solution)
Arsenic trichloride	Butter of arsenic	Yellowish, oily liquid	Pottery
Sodium arsenite	Meta-arsenite, arsenious acid sodium	White or greyish powder	Insecticide, wood preservative, veterinary use
Arsenic pentoxide	Arsenic acid, arsenic anhydride	White powder (dissolves rapidly in water to form arsenic acid)	Manufacture of coloured glass, insecticide, wood presevative
Lead arsenate	Acid lead arsenate	Heavy, white powder	Insecticide
Sulfides of arsenic	Red realgar (disulfide) Yellow orpiment (trisulfide)	Red powder Yellow powder	Depilatory Depilatory and colour pigment
Copper arsenite	Scheele's green	Greenish powder	Colouring agent for toys, wall paper, etc
Copper acetoarsenite	Paris or Emerald green	Greenish powder	Insecticide
Organic arsenicals (carbarsone, tryparsamide, glycobiarsol, melarsoprol, etc)	—	—	Previously used in therapeutics
Arsenic compounds are also used in paints, and the tanning industry			

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Table 4.2 Arsenic Poisoning

System	Acute	Chronic
Dermal	Hair loss, transverse bands of opacity in nails (Mees' lines)	Melanosis (neck, eyelids, nipples), Bowen's disease, facial oedema, hyperkeratosis, pigmentation (rain drop pattern), skin cancer.
Gastrointestinal	Abdominal pain, metallic taste, garlicky breath. Dysphagia, vomiting, bloody or rice-water diarrhoea	Anorexia, nausea, vomiting, diarrhoea, weight loss.
Liver	Fatty degeneration	Hepatomegaly, jaundice, cirrhosis.
Kidney	Oliguria, uraemia.	Nephritic changes
Neurological	Hyperpyrexia, convulsions, coma	Encephalopathy, polyneuritis (glove & stocking type), tremor.
Haematological	——	Anaemia, leucopenia, thrombocytopenia, basophilic stippling, karyorrhexis.
Cardiac	Cardiac arrhythmias	——

Table 4.3 Substances Producing Dermal Pigmentation on Ingestion

Bluish	Brownish	Reddish	Yellowish
Bismuth Mercury Minocycline Oxalic acid Phenothiazines Silver Tetracycline	Arsenic Chromium Levodopa Methyl dopa Nitrates / nitrites Phenacetin	Borates Clofazimine Rifampicin	Carotene Chloroquine Methylenedianiline Nitrazepam Picric acid Quinacrine Sodium nitrate

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4. Radiography: Since arsenic is radiopaque, abdominal x-ray may reveal its presence in the gastrointestinal tract in acute poisoning. Table 4.4 presents a list of radiopaque poisons commonly encountered in practice.

Table 4.4 Radiopaque Poisons

Acetazolamide
Acetylsalicylic acid
Ammonium chloride
Busulfan
Carbon tetrachloride
Chloral hydrate
Chloroform
Enteric coated tablets
Heavy metals
Iodides, iron
Methotrexate
Penicillin G & K
Phenothiazines
Potassium chloride, iodide, & permanganate
Pseudoephedrine
Sodium chloride
Vitamins

Treatment

1. Supportive measures : gastric lavage, intravenous fluids, cardiac monitoring, etc.

2. **Chelation therapy**: This can be done with BAL (British Anti Lewisite or dimercaprol), penicillamine, DMSA (DiMercapto Succinic Acid), or DMPS (DiMercapto Propane Sulfonate). The usual agent employed is BAL at a dose of 3 to 5mg/kg intramuscularly every 4 hours until the urinary arsenic excretion dips below 50mg/24 hours. Usual duration of therapy is 7 to 10 days. In patients who are not allergic to penicillin, penicillamine can be given orally at a dose of 100 mg/kg/day, (6th hourly), for 5 days.

   DMSA and DMPS said to be superior to BAL and penicillamine, are currently not available in India.

3. Haemodialysis or exchange transfusion.

   Note: Chelation therapy is not very effective for chronic poisoning, and is totally ineffective in arsine poisoning. The latter should be treated with emphasis on respiratory stabilisation and haemodialysis.

Autopsy Features

1. Gastrointestinal congestion is a noteworthy feature in acute arsenic poisoning and varies from a mere reddening of mucosa (red velvet) to frank haemorrhagic gastritis. Focal haemorrhages giving rise to a flea bitten appearance is said to be characteristic. The intestines may be inflamed and may contain "rice water" contents
2. Subendocardial haemorrhages are often seen in the heart.
3. There may be evidence of fatty degeneration of heart, liver, and kidneys.

There is a long held belief that arsenic preserves a dead body by delaying putrefaction, though there is not enough scientific validation for this.

Chronic arsenic poisoning may present features of non-specific gastrointestinal inflammation, as well as renal and hepatic damage, at autopsy.

It is conventional to preserve apart from the routine viscera and body fluids, a piece of long bone (preferably femur), a bunch of pulled scalp hair, a wedge of muscle, and a small portion of skin (from the back of the trunk) for chemical analysis. The gruesome practice of pulling out finger and toe nails is best dispensed with.

Forensic Significance

Arsenic has had an outstanding reputation as an ideal homicidal poison especially in the West, part-

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icularly United Kingdom in the Victorian era. Several celebrated murders are said to have been accomplished with the help of arsenic during this period. One of the most shocking cases which remained speculative until recently, when scientific evidence finally established the truth beyond reasonable doubt, was that of Napoleon Bonaparte.

Arsenic trioxide being almost tasteless and colourless in solution can be administered without arousing the suspicion of a victim. The main obstacle is relative insolubility. But the solubility can be greatly enhanced if hot solutions are used such as coffee, tea, cocoa, porridge or gruel, and soups. The only problem is that on cooling, much of the dissolved arsenic will separate out to yield a gritty deposit. On chronic successful administration to a victim, arsenic produces insidious but relentlessly progressive symptoms which are likely to be mistaken even by a medical practitioner for natural causes such as neurological disease, alcoholism, tuberculosis, and hepatic or renal afflictions, while acute poisoning may be confused with gastroenteritis or cholera.

However the popularity of arsenic has declined in recent times because of various factors – legal restrictions on its sale, availability of sophisticated methods which can detect even minute quantities in a dead body, and of course the emergence of better poisons to accomplish nefarious deeds ! Today most cases of arsenic poisoning are accidental, though murders are still reported from time to time.[3] Accidental poisoning may be the result of industrial or occupational exposure, or it may be due to consumption of contaminated water or food. In the past, certain allopathic drugs used to contain arsenic and iatrogenic poisoning was not uncommon. Today ayurvedic preparations are the main hazard, some of which can contain substantial concentrations of arsenic that can produce chronic poisoning on prolonged use.[4]

Outbreaks of arsenic poisoning on a mass scale have been reported from time to time. In 1900, accidental adulteration of beer with arsenic (because of brewing with arsenic-contaminated glucose) left 6000 people poisoned in England, out of which 70 died. Later, in 1943, 150 people were taken ill out of whom 2 died, this time because of consumption of contaminated sausage meat.

Today the most outstanding source of chronic accidental poisoning in India is consumption of well water. There are indications that sizeable populations of several Asian countries are exposed to arsenic tainted water, particularly tube well water.

Lead

*".....sure, tis the lead mills, where the woman gets took on at 18 pence a day, sur... and her constitooshun is lead-poisoned, bad as can be, sur; and her brain is coming out her ear and it hurts her dreadful... ... "**

Lead is the commonest metal involved in chronic poisoning. It was one of the first metals known to man and has been widely used during the last two thousand years for domestic, industrial, and therapeutic purposes. Lead is abundant in soil, being distributed throughout the earth's crust.

Physical Appearance and Uses

Lead is a heavy, soft, steel-grey metal which gives off toxic fumes when melted. Several of its salts occur as variously coloured powders or liquids and are used widely in industry and at home, producing cumulative toxicity on chronic exposure. Lead acetate (sugar of lead) has been used in therapeutics, lead carbonate (white lead) is still used in paints, lead oxide (litharge) is essential for glazing of pottery and enamel ware, and tetraethyl lead is mixed with petrol as an antiknock to prevent detonation in internal combustion engines. Among cosmetics, lead tetroxide is the most common com-

*From "The Uncommercial Traveller" by Charles Dickens (1863).

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pound in vermilion ("sindoor") applied by married Hindu women to the parting of their scalp hair, while lead sulfide is used as a collyrium ("surma") for the eyes by Muslims.

The various situations in which lead salts are used resulting in chronic occupational, environmental, or domesic exposure are listed in Table 4.5.

Usual Fatal Dose

This is not really relevant to lead since acute poisoning is very rare. The average lethal dose is said to be 10 gm/70 kg for most lead salts, while it is 100 mg/kg for tetraethyl lead.

Toxicokinetics

Lead is absorbed through all portals of entry. Occupational exposure results mainly from inhalation, while in most other situations the mode of intake is ingestion. Tetraethyl lead can be absorbed rapidly through intact skin. Following absorption, it is stored in the bones as phosphate and carbonate. Lead is drawn to those areas of the skeleton which are growing most rapidly. These include the radius, tibia, and femur, which are the most metabolically active. The hypermineralisation is reflected in the form of densities which are the classic "**lead lines**" observed on x-ray.

Absorbed lead which is not retained in the body is excreted primarily in the urine (about 65 %) and bile (about 35 %).

Mode of Action

1. Lead combines with sulfhydryl enzymes leading to interference with their action.
2. It decreases haeme synthesis by inactivating the enzymes involved such as aminolaevulinic acid dehydrase, aminolaevulinic acid synthetase, coproporphyrinogen oxidase (or decarboxylase), and ferrochelatase.[5] This results in anaemia.
3. It increases haemolysis as a result of which immature red cells are released into circulation such as reticulocytes and **basophilic stippled cells** (the result of aggregation of ribonucleic acid due to inhibition of the enzyme pyrimidine-5′-nucleotidase which normally eliminates degraded RNA).
4. In the CNS, lead causes oedema and has a direct cytotoxic effect leading to decreased nerve conduction, increased psychomotor activity, lower IQ, and behavioural / learning disorders.
5. Lead also has deleterious effects on the CVS (hypertension and myocarditis), kidney (nephritis), and reproductive organs (infertility). In addition, lead can decrease uric acid renal excretion, thereby raising blood urate levels and predisposing to gout (**saturnine gout**).

Table 4.5 Sources of Lead Exposure

Environmental	Domestic	Occupational
Automobile exhaust *	Ceramic ware +	Autorepair works *
Soil •	Cosmetics •	Battery making*
Water •	House paint*	Glass manufacture *
	Indigenous medicines •	Mining *
	Pencils •	Plastics manufacture *
	Toys •	Plumbing +
		Pottery +
		Printing•
		Rubber industry•
		Smelting & refining*
		Steel welding & cutting*

*High risk + Moderate risk • Low risk

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Permissible blood lead level

Traditionally the accepted upper level for blood lead (BL) has been 80 mg/100 ml, but this was reduced recently to 35 mg/100 ml. However there are reports that adverse effects especially on the haematopoietic system can occur at levels as low as 10 mg/100 ml.[6] Neurobehavioural disorders in children can occur at BL as low as 25 mg/100 ml.[7]

Clinical Features

- **Acute Poisoning**: This is rare. Many reported cases of acute poisoning may actually be exacerbations of chronic lead poisoning when significant quantities of lead are suddenly released into the bloodstream from bone. Symptoms include metallic taste, abdominal pain, constipation or diarrhoea (stools may be blackish due to lead sulfide), vomiting, hyperactivity or lethargy, ataxia, behavioural changes, convulsions, and coma.

- **Chronic Poisoning: (Plumbism or Saturnism)**

  A. Mild Toxicity (BL 40 to 60 mg/100 ml):
  1. Myalgia
  2. Paraesthesia
  3. Fatigue
  4. Irritability
  5. Abdominal discomfort

  B. Moderate Toxicity (BL 60 to 100 mg/100 ml):
  1. Arthralgia (especially nocturnal)
  2. Muscular exhaustibility
  3. Tremor
  4. Headache
  5. Diffuse abdominal pain
  6. Anorexia, metallic taste, vomiting
  7. Constipation
  8. Weight loss

  C. Severe Toxicity (BL more than 100 mg/100 ml):
  1. *Lead palsy* – wrist or foot drop.
  2. A bluish black lead line on gums (*Burton's line*). It is due to deposition of lead sulfide.
  3. *Lead colic* – intermittent severe abdominal cramps. There may be tenderness around the umbilicus.
  4. *Lead encephalopathy* – It is more common in children and is often associated with organic lead toxicity, especially tetraethyl lead or TEL which is lipid soluble and is distributed widely in lyophilic tissues such as the brain. TEL is metabolised to triethyl lead which is the major toxic compound. There is sudden onset of vomiting , irritability, headache, ataxia, vertigo, convulsions, psychotic manifestations, coma, and death. Mortality rate is around 25%. Even if recovery occurs, there is often permanent brain damage manifesting as mental retardation, cerebral palsy, optic neuropathy, hyperkinesis, and periodic convulsions.

  Facial pallor, especially circum-oral is said to be a characteristic feature of chronic lead poisoning and is due to vasospasm, though anaemia may contribute to a significant extent. The anaemia that is encountered in plumbism is similar to that due to iron deficiency i.e., it is hypochromic and microcytic in type ; but anaemia due to the latter is neither associated with reticulocytosis, nor are basophilic stippled cells seen.

Diagnosis: Laboratory tests for lead[8] :

Blood

1. *Complete blood count* and *Peripheral smear* – General and non-specific findings include

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low haematocrit and haemoglobin values with normal total and differential cell counts. The peripheral smear may either be normochromic or hypochromic, and microcytic. Basophilic stippling is usually seen only in patients who have been significantly poisoned for a prolonged period.

2. *FEP* and *ZNp levels* – (> 50 mg/100 ml): An elevated FEP (free erythrocyte protoporphyrin) level indicates impairment of the haeme biosynthetic pathway and may result from lead poisoning or iron deficiency. In order to confirm whether it is due to the former, the BL must be estimated. Today ZnP (zinc protoporphyrin levels) are more commonly studied than FEP.

3. *Blood lead level* (BL): The recommended methods of estimating blood lead level (BL) include atomic absorption spectroscopy (AAS), electrothermal atomic absorption spectroscopy (EAAS), anodic stripping voltametry (ASV), inductively coupled plasma atomic emission spectroscopy (ICP-AES), and x-ray fluorescence spectroscopy.

Urine

1. The concentration of ALA (aminolaevulinic acid) in urine is widely used as a measure of lead toxicity in workers who are exposed occupationally.

2. *Urine lead level*: If this is above 150 mg/litre it is a significant finding, but it is unfortunately not very reliable.

3. *Calcium disodium EDTA mobilisation test*: Procedure – First the patient is asked to empty the bladder. Then $CaNa_2$ EDTA is administered at a dose of 500 mg/m² in 5% dextrose infused over 1 hour. All urine must be collected with lead-free equipment over the next 8 hours. In the laboratory, the urine volume should be carefully measured and stored at 20^0 C until the lead concentration is measured.

To obtain the total lead excreted (mg), the concentration of lead in the urine (mg/ml) is multiplied by total urinary volume (ml). The total urinary excretion of lead (mg) is divided by the amount of $CaNa_2$ EDTA given (mg) to obtain the lead excretion ratio, as follows:

$$\frac{\text{lead excreted (mg)}}{CaNa_2EDTA \text{ given (mg)}}$$

An 8 hour $CaNa_2$ EDTA chelation provocative test is considered positive if the lead excretion ratio is more than 0.6 (though some clinicians use a cut-off of 0.5).

Radiology: This involves evaluation of the ends of long bones for arrest of growth line, and of the abdomen for radiopaque densities. Radiological examination of the abdomen may show radiopaque foreign material if the material has been ingested during the preceding 24 to 36 hours.

The significant finding in bone is the appearance of dense transverse bands or **lead lines** extending across the metaphyses of the long bones, and along margins of flat bones such as the iliac crest. It usually takes 4 to 8 weeks of heavy exposure for dense bands to develop.

Treatment

The Graef Protocol (modified version)[9] –

I. *Severe acute poisoning with encephalopathy*: This is a medical emergency and the following measures must be undertaken immediately
   a) BAL 4 mg/kg immediately (in children).
   b) Cranial CT scan: to rule out cerebral oedema.

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c) KUB: to rule out lead chips in g.i.tract.
d) For seizures : administer diazepam or phenobarbitone.
e) Foley catheterisation : to monitor urinary specificic gravity, sediment, lead level.
f) $CaNa_2$ EDTA 75 mg/kg/day i.v infusion.
g) After the initial dose of BAL, repeat the same dose at 4 hourly intervals until blood lead level falls below 40 mg/100 ml. Then reduce BAL to 12 mg/kg/day in 3 divided doses.
h) Reduce $CaNa_2$ EDTA to 50 mg/kg/day as condition improves.
i) Continue the above regimen until patient is asymptomatic and can tolerate oral chelation with D-penicillamine or DMSA.

II. *Severe poisoning without encephalopathy* (BL more than 70 mg/100 ml) :
a) BAL 12/mg/kg/day.
b) EDTA 50 mg/kg/day.
c) Discontinue BAL when the BL falls below 40 mg/100 ml, but continue EDTA for 5 more days.
d) Change to oral chelation subsequently which may have to be continued until the BL falls below 15 mg/100 ml, or 3 months have been completed.

III. *Moderate poisoning* (BL between 45 and 70 mg/100 ml) :
1. EDTA 50 mg/kg/day.
2. When blood lead falls below 40 mg/100ml, begin oral chelation.

IV. *Mild poisoning* (BL 20 to 35 mg/100 ml): D-Penicillamine 30 mg/kg/day in 3 divided doses. Start with 1/4th of the calculated dose. Double this after 1 week. Double again (to full dose) after 1 week. Continue this until the BL falls to less than 15 mg/100 ml, or 3 months have been completed.

In addition to the (modified) Graef protocol , the following supportive measures must be instituted as applicable –
1. Thiamine 10 to 50 mg/kg is said to improve neurological manifestations of lead poisoning.[10]
2. In acute poisoning, or in the event of radiopacities in the g.i.tract on x-ray, stomach wash can be done.
3. Lead colic usually responds to i.v calcium gluconate.

In recent times, a new chelating agent called Succimer i.e., DMSA has been introduced in the management of lead poisoning in western countries. It is said to be more efficacious and less toxic.

Autopsy Features
1. Pale skin, conjunctivae, and mucosa.
2. Emaciation.
3. Burtonian line.
4. Lead lines on x-ray.

Forensic Significance
Metallic lead has been part of the human environment for over 5000 years, and is today detectable in practically all phases of the inert environment and in all biological systems worldwide. Because of its malleability and low melting point, it was one of the first metals smelted and used by early human societies. Hippocrates is credited with the earliest description of chronic lead poisoning when he associated persistent abdominal colic in a man, with his occupation of extracting metals (around 370 BC). The ancient Romans were however the first to experience the metal's adverse effects on a massive scale mainly because of chronic poisoning through lead acetate which was used to sweeten wine in those days.

More recently, lead-laced wine and lead-based cosmetics caused havoc among the English in the 18th and 19th centuries which included even members of the royalty. The many ailments suffered

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by Queen Anne including her disastrous obstetric history are said to be because of lead poisoning.[11] In 1763, Benjamin Franklin reported on dry gripes (abdominal colic), and dangles (wrist drop), which afflicted painters and typesetters, as well as the gripes due to rum distillation in leaden condensing coils. In 1863, Charles Dickens described graphically the plight of lead mill workers in "The Uncommercial Traveller".

Today chronic lead poisoning is said to be the most important environmental health problem, particularly among young children.[12] Children are especially susceptible to lead poisoning because of their increased absorption of lead from the g.i.t when compared with the adult. The child with pica is in enhanced danger because of the tendency in such a case to lick lead-based paints off walls, furniture, toys, pencils, etc. Sadly, a child's environment (particularly in third world countries such as India) is full of lead. Exposure can occur through paint, petrol, soil, food, water, and even air. Millions of children are probably affected annually leading to permanent neurological sequelae. The increasing antisocial behaviour of street urchins and slum dwelling children in congested and polluted cities may also be the offshoot of chronic lead poisoning, since studies have demonstrated a high incidence of plumbism in juvenile boys with delinquent behaviour.[13] In addition, the alarming rise in recent years of learning disabilities, lowered IQ, and memory problems of children may be related to the effects of plumbism. A Pilot Study undertaken by a charitable trust in major cities of India in 1998 has disclosed significant blood lead levels in school and slum children.

Recent studies have also revealed significant lead levels among petrol pump and garage workers many of whom display varying degrees of clinical manifestations.[14] Today unleaded petrol has finally made its entry into the country, and while the situation is expected to improve in the coming years, presently only a few cities have petrol pumps dispensing lead-free petrol.

The most sizeable chunk of plumbism occurs from occupational exposure among workers exposed to lead such as miners, plumbers, battery makers, plastic manufacturers, garage workers, etc.

As would be evident from the foregoing, lead poisoning is almost always accidental or inadvertent in nature. Instances of suicide or homicide with lead-based compounds are extremely rare.

Mercury

Synonyms

Quicksilver

Physical Appearance

Metallic or elemental mercury (Hg^0) is a heavy, silvery liquid which is per se non-toxic, but vapourises at room temperature to give off a toxic vapour : mercuric mercury.

Salts or compounds of mercury may be inorganic or organic in nature. Inorganic salts are of two types – *mercuric* (bivalent, i.e., Hg^{++}) and *mercurous* (monovalent, i.e., Hg^{+}). Common mercuric salts include mercuric chloride (corrosive sublimate) which is a white crystalline corrosive powder, mercuric oxide which is a red crystalline powder that turns yellow when treated with caustic soda or potash, and mercuric sulfide (vermilion or sindoor) which is a red crystalline powder. The most important mercurous salt is mercurous chloride (calomel) which is fibrous and heavy.

Organic mercurials are generally more toxic and comprise mainly compounds such as phenyl and methoxymethyl mercury, and alkyl compounds such as ethyl and methyl mercury. Mercurochrome is also an organic mercurial and exists as iridescent green scales readily soluble in water. The most toxic compound of mercury is methyl mercury.

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Table 4.6 Uses of Mercury Compounds

Industry	Medicine & Dentistry	Miscellaneous
1. Barometer, thermometer, etc 2. Ceramics 3. Dry cell batteries 4 Electrical appliances (mercury switches) 5. Explosives & fireworks 6. Felt hats 7. Fluorescent & mercury vapour lamps	1. Antiseptic & disinfectant 2. Dental amalgam 3. Diuretic 4. Purgative	1. Electroplating 2. Embalming 3. Fabric softener 4. Fingerprint powder 5. Fungicide 6. Gold & silver extraction 7. Grain preservative 8. Paints 9. Pesticides

Uses

See Table 4.6.

Usual Fatal Dose

Mercuric chloride : 0.5 to 1 gm/70 kg

Mercurous chloride : 1.5 to 2 gm/70 kg

Toxicokinetics

After inhalation, elemental mercury is readily absorbed through the alveolar membrane and enters the blood stream. Ingestion of mercury salts is associated with slower rate of absorption. Mercury is rapidly converted to mercuric ions (Hg^{++}) in the blood which can lead to renal tubular damage during excretion. In the central nervous system, mercury acts mainly upon cerebellum, temporal lobe, basal ganglia, and corpus callosum.[15]

Both organic and inorganic mercurials can be absorbed through intact skin.

Clinical Features

I. **Poisoning with elemental mercury and inorganic salts:**

- **Acute Poisoning:**
  - *Inhalation*: This usually occurs while heating metal in a closed room, or following gold refining in an enclosed area. Symptoms (which may be delayed upto 4 hrs) include dyspnoea, cough, fever, headache, chills*, g.i disturbances, metallic taste, and blurring of vision. In severe cases there may be non-cardiogenic pulmonary oedema, dyspnoea, convulsions, etc. Sometimes manifestations similar to *Kawasaki disease* (mucocutaneous lymph node syndrome) are seen especially in children, which may be mistaken for scarlet fever: conjunctival congestion, fever, reddened palms and soles, deep red oral mucosa with "strawberry tongue", skin rash, and cervical lymphadenopathy.
  - *Ingestion*: Small quantities of elemental mercury usually cause no harm on ingestion. Sometimes even a relatively large amount may pass out of the body uneventfully (egged on by a mild laxative!). It is however advisable to take x-rays to monitor the progress of the elemental mercury through the g.i tract.

Ingestion of mercuric salts produces corrosion leading to abdominal pain, vomiting, diarrhoea, and shock . The

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* Metal fume fever (see page no. 335)

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mucosa of the g.i tract usually appears greyish. There may be haematemesis. In severe cases there is onset of renal failure, pulmonary oedema, and coma. Urine may appear pinkish. Ingestion of button (or disc) batteries poses special problems since they contain a variety of caustic substances apart from mercury.

- *Injection*: Subcutaneous or intramuscular injections of elemental mercury may cause abscess formation wih ulceration, extruding tiny droplets of mercury. Intravenous injection* can result in **mercurialism** characterised by thrombophlebitis, granuloma formation, and pulmonary embolism. Repeated haemoptysis is a characteristic feature.

- **Chronic Poisoning** (*Hydrargyrism*) – *Inhalation*

1. Tremor : It is one of the classical and most consistent manifestations of chronic mercury poisoning and is sometimes referred to as the **Danbury tremor**. It begins in the hands and is of a coarse, intentional type, interspersed with jerky movements. Later it progresses to the lips, tongue, arms, and legs. The advanced condition is referred to as **Hatter's shakes**, when the tremor becomes so severe that daily activities involving some delicacy of movement become grossly impaired, for e.g., shaving, writing, holding a tumbler or spoon, etc. The most severe form of the condition is referred to as **concussio mercurialis** when literally no activity is possible. Even years after exposure to mercury has ceased, tremor may persist.
2. Ataxia, reeling gait.
3. Metallic taste (Table 4.7), anorexia, nausea, increased salivation.

Table 4.7 Poisons/Drugs Producing Metallic Taste

Acetaldehyde	Iodine
Allopurinol	Iron salts
Arsenicals	Lead
Cadmium	Lithium
Copper	Mercury
Disulfiram	Metoclopramide
Ethambutol	Metronidazole

4. Gingivitis, halitosis, blue line on gums (similar to the Burtonian line of plumbism).
5. **Erethism** : Another classic manifestation, it refers to a cluster of psychiatric symptoms including abnormal shyness, loss of self confidence, depression, irritability, amnesia, excitability, progressing in later stages to delirium with hallucinations, or suicidal melancholia, or manic depressive psychosis (**mad hatter**).
6. **Mercuria lentis** : Characterised by the brown reflex of anterior lens capsule of the eye. There may be fine punctate opacities.

** May be a deliberate suicidal gesture, or the result of a misconception that such injections can enhance athletic or sexual performance. In fact the term "quicksilver" has its origins in the false belief that mercury can quicken a boxer's punches.*

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7. Renal damage results in membranous glomerulonephritis with hyaline casts and fatty casts in the urine.

- *Ingestion*
  1. Colitis.
  2. Melanosis coli.
  3. Dementia.
  4. Tremor.
  5. Renal failure.
  6. **Acrodynia (Pink disease)** : This is seen mainly in children. Formerly most cases were related to chronic use of teething powders containing mercurous chloride. But almost any form of chronic mercury exposure can cause this condition including inhalation of elemental mercury vapour or cutaneous application of ammoniated mercury ointments. The onset is usually insidious with anorexia, insomnia, profuse sweating, skin rash, and photophobia. The hands and feet become puffy, pinkish, painful, paraesthetic, perspiring, and peeling (*the child pees!*). Teeth may be shed, with ulceration of gums.

II. **Poisoning with organic mercurials:** Features are related mainly to the CNS and include dysarthria, ataxia, parasthesias, neuropathies, diminished auditory and visual acuity, mental deterioration, and chorea.

A special type of organic mercurial poisoning relates to food (fish) poisoning through methyl mercury which has caused terrible tragedies in the past (**Minimata disease**), and may well cause more havoc in the years to come, unless measures are devised to curb its far reaching effects.

Diagnosis

1. *X-ray*
2. *Blood mercury level* : Flameless atomic absorption spectrometry is best for deducing this. Normal level is less than 3 mg/100 ml.
3. *Urine mercury level* : Normal level is less than 10 to 15 mg/100 ml.
4. *Hair analysis*: Done by cold vapour atomic absorption spectrometry. External contamination can however vitiate the results.

Treatment

Acute Poisoning

I. Metallic mercury and inorganic compounds:

A. *Inhalation*
   1. Removal from source of exposure.
   2. Supportive measures.
   3. Chelation (**Table 4.8**).

B. *Ingestion*
   1. In elemental mercury ingestion, take x-ray and repeat it to study the progression.
   2. Administer laxatives.
   3. Demulcents for corrosive compounds such as mercuric chloride.
   4. Stomach wash : It may be advisable to add egg white or 5% albumin or just plain milk to the lavage fluid to bind the mercury.
   5. Chelation.

C. *Injection*
   1. If there is abscess formation, perform repeated incisions to remove the mercury.
   2. Monitor the CNS and renal functions for evidence of toxicity.
   3. Mercuric salts are relatively well adsorbed by activated charcoal.
   4. Chelation.

II. Organic Mercurials:
   1. Supportive measures.
   2. Chelation is not very effective.

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Chronic Poisoning: The mainstay of chronic mercury poisoning is chelation therapy (**Table 4.8**).

Table 4.8 Chelation Therapy for Mercury Poisoning

1. **BAL** (British Anti Lewisite)
   100 mg by deep i.m., every 4 hrs for 48 hrs, followed by 100 mg every 8 hrs for 8 to 10 days.

   OR
2. **DMPS** (2,3 DiMercaptoPropane-1-Sulfonate)
   5 mg/kg i.v., or 6 infusions of 250 mg/day, followed by 100 mg orally twice a day for 24 days.

   OR
3. **DMSA** (Meso 2,3 DiMercaptoSuccinicAcid, or Succimer)
   30 mg/kg/day orally for 5 days, followed by 20 mg/day for 14 days.

   OR
4. **D-Penicillamine**
   250 mg q.id., for adults, (20 mg/kg/day)

Autopsy Features

In death due to acute mercury poisoning, the mucosa of the mouth, throat, oesophagus, and stomach appears greyish in colour with softening and superficial corrosion. There may be haemorrhagic points. If there had been survival for a few days, the large intestine may reveal ulceration. Kidneys are often pale and swollen due to oedema of renal cortex. Microscopy usually demonstrates necrotic changes in the renal tubules.

Forensic Significance

Mercury and its compounds constitute important industrial, agricultural, and occupational toxic hazards. In the past when mercury was used in its various forms for the therapy of a wide variety of ailments including syphilis, iatrogenic poisoning was common. Even today calomel (mercurous chloride) is sometimes recommended as a laxative. Prolonged use can cause colitis, dementia, tremor, and renal failure. Acrodynia resulting from teething powders containing calomel has fortunately become a rarity.

The tragic consequences of chronic industrial exposure to mercury have been outlined in the foregoing sections.

Domestic exposure though not often reported is today a distinct threat. Such exposure may result from inadvertent mercury spills (broken thermometers), or from home gold-ore processing.

An important area that must be considered in relation to chronic mercury exposure is dentistry. Dental amalgams used for fillings often contain mercury. Dental surgeons may get chronically exposed due to this reason. However patients with mercury amalgam fillings are not at risk even though such fillings may remain intact for years.[16]

Food poisoning (especially involving fish) through mercury compounds is not uncommon.

A relatively new hazard concerns dry cell batteries used in cameras, hearing aids, torches, and watches. These are small metal capsules ranging in size from 7 to 25 mm in diameter and 0.5 cm thick. Each contains about 2 gm mercuric oxide interleaved with zinc amalgam. The capsule is usually made of stainless steel and is not likely to be corroded by gastric juice if the battery is swallowed. However this sometimes happens and can (theoretically) release toxic levels of mercury. All such cases should therefore be monitored carefully with serial x-ray evaluations. Surgical intervention is usually not necessary and the battery is excreted in due course in the faeces. A mild laxative can hasten its passage.

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Iron

Physical Appearance

Metallic iron is silvery white in colour, occurring naturally as haematite, magnetite, etc., and usually causes no problems. In fact it is an essential element and deficiency results in anaemia. Even if there is more than the required intake daily, the excess is excreted. But in some individuals with inborn errors, even normal dietary iron can cause toxic effects due to accumulation e.g., **haemochromatosis (bronze diabetes).**

Various iron salts are administered therapeutically in individuals with iron deficiency anaemia which can result from a wide variety of causes. Iron poisoning is related in most instances to overdose of such salts. One of the commonest is ferrous sulfate (green vitriol) which occurs as bluish green crystals.

Uses

Medicinal:

1. Treatment of iron deficiency anaemia.
2. Astringent.

Industry: Tanning, inks, dyes, pigments.

General: Photography.

Usual Fatal Dose

Commonly used iron salts in therapeutics along with respective iron content are mentioned in **Table 4.9**

The usual fatal dose corresponds to about 200 to 250 mg of elemental iron per kg of body weight. This can be calculated from the percentage of elemental iron in a particular preparation for e.g., a single 150 mg tablet of anhydrous ferrous sulfate which contains 37% of elemental iron, will contain a total of 55 mg of elemental iron. But such calculations can be misleading since serious hepatotoxicity can result at much lower concentrations of iron in the body which can lead to death. In practice, this can be as low as 60 mg of elemental iron/kg. Hence just a handful of these tablets (15 to 20 in number), can be lethal to a young child.

Toxicokinetics

Iron poisoning occurs when serum iron level exceeds the total iron-binding capacity (TIBC), resulting in free circulating iron in the bloodstream.

Mode of Action

Free iron causes

1. Massive postarteriolar dilatation which results in venous pooling.
2. Increased capillary permeability resulting in decreased plasma volume.
3. Oxidation of ferrous to ferric iron releasing hydrogen ions. Subsequent hydration of ferric iron results in metabolic acidosis.
4. Inhibition of mitochondrial function leading to hepatic damage, hypoglycaemia, and hypoprothrombinaemia.
5. Direct corrosive action on the g.i mucosa.

Table 4.9 Elemental Iron Content of Iron Salts

Iron Salt	Iron Content (%)	Iron Salt	Iron Content (%)
Ferrous gluconate	12	Ferrous fumarate	33
Ferric pyrophosphate	12	Ferric chloride (anhydrous)	34
Ferric ammonium citrate	14 to 18	Ferrous succinate	35
Ferroglycerine sulfate	16	Ferrous sulfate (anhydrous)	37
Ferrous sulfate(hydrated)	20	Ferric phosphate	37
Ferric chloride(hydrated)	20	Ferrous chloride(anhydrous)	44
Ferrous chloride(hydrated)	28	Ferrous carbonate	48

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Clinical Features

Most cases occur in children. There are 4 stages.

Stage I (30 min to 6 hrs post-ingestion):
Vomiting, diarrhoea, abdominal pain, hypotension, pallor, lethargy, significant anion gap metabolic acidosis, leucocytosis, and hypoglycaemia.

Stage II (6 to 24 hrs):
Hypovolaemia, lethargy, hypotension, and severe metabolic acidosis. The child may appear to be feeling better, which is actually treacherous since the III stage begins and unfolds in the form of a relentless Greek tragedy.

Stage III (24 to 48 hrs): There is multiple organ failure involving the gastrointestinal, hepatorenal, central nervous, and cardiovascular systems, with metabolic coagulopathies, hypoglycaemia, convulsions, disorientation, coma, and death (usually from hepatic failure).

Stage IV (After 4 weeks): Recovery is often associated with late complications such as gastric scarring and pyloric obstruction resulting from corrosion of g.i mucosa.

Diagnosis

1. *X-ray*: Like all other heavy metals, iron and its compounds are radiopaque. However, chewable iron tablets and liquid iron formulations are usually not visualised on x-ray.
2. *Serum iron level*: Poisoning is indicated if this exceeds 150 mg/100 ml, and serious toxicity is usually associated with levels beyond 500 to 600 mg/100 ml.
3. *Chelation challenge test*:Desferrioxamine in a dose of 25mg/kg (maximum 1 gm) is given i.m. If the serum iron has exceeded iron binding capacity, the excess iron is chelated to desferrioxamine and the complex is excreted as a pinkish (Vin Rose) colour in the urine. But a negative result does not rule out iron poisoning.
4. *Qualitative desferrioxamine colour test (QDCT)* : 2 ml of gastric fluid and 2 drops of 30% hydrogen peroxide are placed in 2 plastic tubes. 0.5 ml of solution of desferrioxamine (500 mg in 4 ml distilled water) is added into one tube and the resulting colour change is compared with the other tube (control). If the test is positive, an orange to red colour will develop in the tube in which desferrioxamine was added. The test must be done within 2 hours of ingestion of iron.

Treatment

1. Stomach wash with normal saline performed gently may be of benefit in massive ingestions.
2. Magnesium hydroxide solution (1%) administered orally may help reduce absorption of iron by precipitating the formation of ferrous hydroxide.
3. Correction of hypovolaemia, and metabolic acidosis.
4. **Chelation therapy**: Chelation can be done either with desferrioxamine (parenteral) or deferiprone (oral). In India, desferrioxamine is available as 500 mg vials for parenteral administration. Addition of 2 ml of sterile injection water results in a solution of 250 mg/ml. The recommended dose is 1 gm i.m (in adults), followed by 500 mg 4th hourly for 2 doses, and finally 500 mg 4th to 12th hourly upto a maximum of 6 gm in 24 hours. It can also be given as an i.v infusion (15 mg/kg/hr) in normal saline, glucose in water, or Ringer's lactate, and this is the preferred method in children.
5. Liver transplantation is the only therapeutic avenue open in the presence of fulminant hepatic failure.

Autopsy Features

1. Haemorrhagic necrosis of gastric mucosa. In ferrous sulfate poisoning, gastric contents may appear bluish green in colour.
2. Hepatic and renal necrosis

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Forensic Significance

Acute iron poisoning has assumed grave significance in recent years and cases of accidental poisoning are being reported with alarming frequency in young children. Since most iron preparations (syrups and tablets) are brightly coloured and pleasantly flavoured, they constitute an irresistible, fatal attraction for these innocent victims. Introduction of childproof containers would be very effective in minimising inadvertent ingestions by children as demonstrated by the Western experience.

Copper

Physical Appearance

Copper is a brown coloured metal and is not toxic per se. In fact it is a normal constituent of the human body. But several of its salts are poisonous, most of which are crystalline in nature and greenish in colour, except for copper sulfate which is bluish (Table 4.10).

Uses

1. Insecticide.
2. Fungicide.
3. Algicide (to destroy algae in swimming pools).
4. Alloys : for e.g., brass (with zinc), bronze (with tin), and money (with nickel).
5. Pigments : for e.g., copper chromate, red copper oxide, etc.

Table 4.10 Toxic Salts of Copper

Chemical Name	Common Name
Copper acetoarsenite	Paris green
Copper arsenite	Scheele's green
Copper carbonate (native)	Mountain green
Copper carbonate (with chalk)	Brunswick green
Copper subacetate	Verdigris
Copper sulfate	Blue vitriol

Usual Fatal Dose

10 to 20 gm of copper sulfate.

Toxicokinetics

The safe daily intake of dietary copper is 2 to 3 mg/day, while the actual requirement is only 0.8 mg/day.[17] It is required for the functioning of enzymes such as catalase and peroxidase.

Copper is normally present in serum in two forms – one which is bound to albumin (7% of total serum copper), and one which is bound to the copper enzyme caeruloplasmin (93%). Total body copper content is 150 mg.

Clinical Features

Acute Poisoning: Copper intoxication is characterised by myalgia, abdominal pain, vomiting (bluish or greenish), diarrhoea, acidosis, pancreatitis, methaemoglobinaemia, haemolysis, jaundice, oliguria, renal failure, seizures, delirium, and coma.

Chronic Poisoning: Chronic inhalation of copper sulfate spray used as an insecticide in vineyards can cause *vineyard sprayer's lung disease* characterised by a histiocytic granulomatous lung. Liver damage is also common.

Chronic contact with swimming pool water containing algicidal copper chemicals can cause green hair discolouration.[18]

Churches in some developing countries distribute "spiritual green water" to devotees which contains copper sulfate. When this is ingested, serious toxicity and even death can result.[19]

Cooking in copper or brass vessels can cause copper poisoning due to verdigris. Copper poisoning can also result from the leaching of copper containers in which carbonated water, citrus fruit juices, and other acidic beverages have been stored

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Chronic copper toxicity is the hallmark of Wilson's disease, an autosomal recessive genetic disorder in which there is deficiency of caeruloplasmin.

Diagnosis

1. *Serum caeruloplasmin level*: A value of 35 mg% or less at 24 hours is associated with serious toxicity.
2. *Blood copper level*: If this is elevated beyond 1.5 mg/100 ml, there is likelihood of serious toxicity.

Treatment

1. Haemodialysis is said to be useful in the early stages of poisoning when the metal is still circulating in the blood stream as free copper.
2. Administration of egg white or milk orally may help in detoxifying copper resulting in the formation of an albuminate.
3. Stomach wash can be done with a solution of potassium ferrocyanide. It converts the copper salt (especially copper sulfate) into insoluble cupric ferrocyanide.
4. Induction of emesis is contraindicated.
5. In severe cases, chelation can be done initially using dimercaprol 2.5 mg/kg, 4th hourly i.m, followed by oral penicillamine in a dose of 2 gm/day

Autopsy Features

1. Jaundiced skin and conjunctivae.
2. Greenish or bluish stomach contents.
3. Hepatic and renal necrosis.

Forensic Significance

In India, the incidence of suicidal ingestion of copper sulfate was quite high till recently. Of late there has been a decline. One study revealed that while copper sulfate accounted for nearly 13% of poisoning cases in the 1970s, this fell to less than 5% in the 1980s.[20] A recent survey shows this to be only around 1% of the total.[21]

Metal Fume Fever

Synonyms

Brass chills, brazier disease, foundry fever, Monday fever, smelter shakes, welder's ague.

Occupations Affected

Welding, galvanising, smelting, metal refining, electroplating, alloy making, ship breaking, etc.

Metals Involved

The syndrome is caused by inhalation of fumes produced when the following metals are heated above their melting point (in decreasing order of importance) : zinc, copper, magnesium, iron, chromium, cadmium, nickel, manganese, mercury, cobalt, lead, antimony, selenium, beryllium, vanadium, silver, and aluminium.

Clinical Presentation

The syndrome resembles a flu-like illness, beginning 4 to 6 hours after exposure to fumes, and is characterised by chills, fever, myalgia, cough, dyspnoea, fatigue, metallic taste, salivation, thirst, sweating, tachycardia, tachypnoea, leukocytosis, cyanosis, and reduced pulmonary function tests. Resolution of symptoms occurs 36 hours after stoppage of exposure, so that over the weekend usually the worker becomes allright only to succumb once again the following Monday (*Monday fever*).

Treatment

Symptomatic and non-specific. Chelation therapy may be required.

Prevention

1. Implementation of engineering controls.
2. General room ventilation.
3. Local exhaust ventilation.
4. Down-draft or cross-draft tables.
5. Use of fume extractors.

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Related Syndromes

A condition very closely resembling metal fume fever called polymer fume fever results from inhalation of gases produced by burning of polytetrafluoroethylene (Teflon).

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CHAPTER - 5

Irritant plants

The blood-like blossom, the dull red fruit,
Though the heart yet sickens, the lips yet sweet,
With sullen savour of poisonous pain.

Swinburne

There are numerous plants which qualify as "irritants", and it would not be possible to deal with them all in a book of this size. Only the common culprits are discussed. Most of these plants have toxic parts (leaf, fruit, seed, bark, root, etc.), which when ingested cause gastrointestinal distress. There may also be involvement of other organ systems, but this is usually not significant.

Ricinus communis

Common Name
Castor, Mole bean

Family
Euphorbiaceae

Characteristics
Large shrub with greenish-red leaves. Fruits are borne in clusters and are soft-spined brown capsules with 2 to 3 mottled grey-brown seeds.

Active Principles
Seed is the toxic part and yields a pale yellow oil with a faint odour and acrid taste. It contains **ricinoleic acid**. The press cake left behind after extraction of oil contains a *toxalbumen* (toxic protein which causes haemolysis) called **ricin**. It is classed as a "super toxic poison", and is said to be even more poisonous than cobra venom.[1]

Uses
Castor oil is used as
- Purgative.
- Industrial lubricant (impure oil).
- Quack remedy for assorted ailments.

Clinical Features
- Vomiting, diarrhoea, abdominal pain.
- Hypotension, dehydration.
- Fever with chills.
- Haemolysis, renal failure.

Usual Fatal Dose
1 mg/kg body weight of ricin; 5 to 10 seeds.

Treatment
- Decontamination (gastric lavage, activated charcoal).
- Correction of fluid and electrolyte imbalance.
- Supportive measures.

Autopsy Features
- Gastrointestinal congestion, erosions.
- Renal congestion.

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Forensic Significance
- Accidental ingestion (children).
- Iatrogenic overdose of castor oil.
- Homicidal (see **Box 5.1**).

Croton tiglium
Family
Euphorbiaceae

Characteristics
Evergreen tree with smooth ash-coloured bark, ovate-lanceolate leaves, small flowers, and oblong, three-lobed fruits containing oval, dark brown seeds. Oil extracted from the seeds is extremely toxic.

Active Principles
- Crotin (toxalbumen)
- Crotonoside (glycoside)

Clinical Features
Similar to castor seed poisoning. Contact of oil with skin causes inflammation.

Usual Fatal Dose
1 to 2 ml of oil; 5 to 6 seeds.

Treatment
As for castor.

Forensic Significance
- Accidental ingestion, usually arising out of mistaken identity with castor oil or seed.
- Oil is used as an abortifacient in rural areas.

Euphorbia pulcherrima (Poinsettia)
Common Name
Christmas plant

Family
Euphorbiaceae

Characteristics
This plant yields a latex-like sap which is a potent irritant. Leaves are large with serrated margins, and turn red, yellow, or pink in the fall.

Active Principle
Diterpene (an ester)

Clinical Features
- Oral burning.
- Vomiting, diarrhoea, abdominal cramps.
- Contact dermatitis.

Treatment
- Gastric lavage
- Antacids
- Wash affected skin with soap and water.

Calotropis gigantea
Family
Asclepiadaceae

Characteristics
Tall shrub with yellowish-white bark, oblong thick leaves, purplish or white flowers, and small ovate flattened seeds, (**Fig 5.1**). Stem and leaves when incised or crushed yield a whitish, milky, acrid juice.

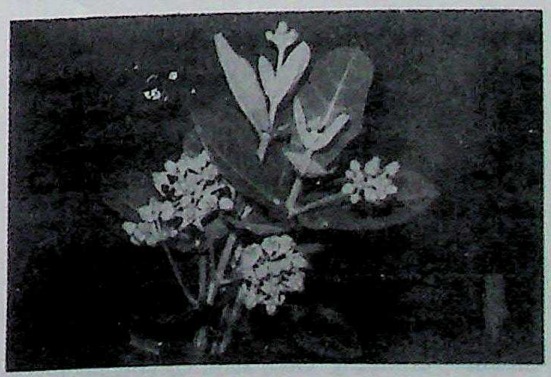

Fig 5.1 Calotropis

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Active Principles
- *Calotoxin*
- *Calotropin*
- *Calactin*
- *Uscharin*

Uses
Various parts of the plant or its juice are used in folk remedies for assorted ailments.

Clinical Features
- Vomiting, diarrhoea, abdominal pain.
- Mydriasis, convulsions, delirium.
- Skin contact with the juice causes inflammation and vesication.
- Eye contact with the juice causes severe conjunctivitis.

Treatment
- Gastric lavage
- Demulcents
- I.v fluids
- Diazepam for convulsions.
- Wash skin lesions with soap and water; eye exposure to be managed with saline irrigation.

Forensic Significance
- Juice is used as an abortifacient.
- *Malingering*: production of artificial bruise or conjunctivitis with the juice.
- Accidental poisoning due to quackery.
- Cattle poison.

Abrus precatorius

Common Names
Jequirity, Rosary bead, Indian liquorice

Family
Leguminoceae

Characteristics
Slender, climbing vine, with compound leaves having 10 to 15 pairs of narrow leaflets, small pinkish flowers, and 4 to 6 seeds inside seed pods which split open when ripe. The seeds are usually bright scarlet in colour with a black spot on one side, measuring 1 cm in size on an average.

Active Principles
Seeds contain
- *Abrin* (toxalbumen)
- *Abrine* (amino acid)
- *Abralin* (glucoside)
- *Abric acid*

Uses[2]
- Seeds are often used in rosary beads, necklaces, etc., by rural folk.
- Jewellers in India sometimes use the seeds as a measure for weighing gold or precious stones

Clinical Manifestations
- Ingestion of seeds or extract can cause haemorrhagic gastritis.
- Injection of extract can cause cardiovascular manifestations akin to a viperine bite. Cardiac arrhythmias, convulsions, and cerebral oedema have been reported.[3]
- Death is not uncommon.

Usual Fatal Dose
90 to 120 mg of abrin ; 1 to 2 seeds. Abrin (like ricin) is a "super toxic" poison.

Treatment
- Decontamination
- Supportive measures.

Autopsy Features
- Congestion of g.i tract and other viscera.
- If injected, local signs of inflammation are seen

Forensic Significance
- Accidental poisoning is not uncommon among children who chew on the attractive seeds out of curiosity.

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- The extract of the seeds is used in rural India to kill cattle, by injecting needles ("sui" in Hindi) made out of the dried seed paste.
- Homicides have been reported with "suis".[4]

Semecarpus anacardium

Common Name
Marking nut

Family
Anacardiaceae

Characteristics
It is a tree bearing hard nut-like fruits, the acid blackish juice of which is used by washermen (dhobis) to mark clothes (and hence the common name – "marking nut"; Fig 5.2).

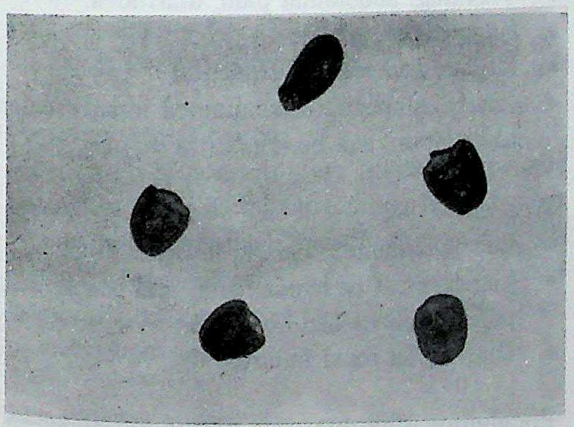

Fig 5.2 Semecarpus Anacardium

Active Principles
The juice contains
- *Semecarpol*
- *Bhilawanol*

Uses
- Laundry marker.
- Quack remedy for assorted ailments.

Clinical Manifestations
- Skin contact with the juice results in inflammation and vesication.
- Ingestion causes gastrointestinal distress, hypotension, and delirium.

Usual Fatal Dose
10 gm

Treatment
Supportive measures.

Forensic Significance
- Juice is used as an abortifacient.
- *Malingering*: production of artificial bruise or conjunctivitis with the juice.
- Accidental poisoning due to quackery.

Capsicum annuum

Common Name
Chilly, Red pepper, Cayenne pepper

Family
Solanaceae

Characteristics
It is a small herb bearing longish tapering fruits which become red when ripe, and possess a pungent odour and taste. The fruit (chilly) contains a number of small, flat, yellowish seeds which bear a superficial resemblance to datura seeds, (Table 5.1 mentions differentiating points).

Active Principle
Capsaicin (alkaloid)

Uses
- Fruit and seeds are popular in Indian cuisine as condiment/flavouring agent. Also used in pickles and sauces.
- Carminative, appetite stimulant.
- Counterirritant in balms.

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Table 5.1 Differences between seeds of chilly and datura

	Chilly	Datura
Size	Small	Large
Colour	Yellow	Brown
Shape	Rounded	Reniform (kidney shaped)
Surface	Smooth	Pitted
Odour	Pungent	Odourless
Taste	Pungent	Bitter
Cut-section	Embryo curves inwards towards the hilum	Embryo curves outwards

Clinical Features

- Skin contact results in irritation and reddening; eye contact causes intense burning, lacrimation, and reddening.
- Ingestion of large quantities causes burning sensation in the mouth, salivation, abdominal pain, vomiting, and diarrhoea. Sweating is common. Urine may turn dark.

Treatment

- Bathe affected skin in vinegar or ice cold water.[5]
- In the case of ingestion, give ice cubes to suck, or sips of ice cold water.
- Supportive measures.

Forensic Significance

- Workers in pickle manufacturing units often suffer from dermatitis and burning lesions of fingers (*Hunan hand*).[6]
- Robbery, rape, etc., may be facilitated by rendering the victim in an agonised and helpless state by throwing powdered chilly in the eyes.
- The powder may also be used for torture or extortion, by forcing it into rectum or vagina.
- Datura seeds may be mistaken for chilly and eaten, giving rise to serious poisoning (page no. 401).

Colchicum autumnale

Family

Colchicaceae

Characteristics

It is an annual herb with narrow leaves (which broaden slightly at the tip), large yellow flowers, and brownish conical corns.

Active Principles

- *Colchicine*
- *Demecolcin*

Clinical Manifestations

- Vomiting, abdominal pain, diarrhoea.
- Hypotension, shock.
- Hepatic and renal dysfunction.
- Rarely, alopecia, disseminated intravascular coagulation, and bone marrow failure.

Treatment

- Decontamination: gastric lavage.
- Management of hypotension and shock with pressor amines and i.v fluids.
- Dialysis for renal failure.

Eucalyptus globulus

Common Name

Eucalyptus, Blue gum

Characteristics

It is a tall tree that grows well in the Nilgiris, and has a smooth bark, long curved leaves, and large flowers.

Active Principle

Eucalyptus oil is obtained by steam distillation of an extract derived from the leaves. The main active principle is *eucalyptol (cineole)*.

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Uses
Eucalyptus oil is used as
- Ingredient of pain balms.
- Remedy for common cold.

Clinical Features
- Burning pain in the mouth, vomiting, diarrhoea, abdominal pain.
- Bronchospasm, tachypnoea, chemical pneumonitis, respiratory depression.
- Headache, vertigo, drowsiness, slurred speech, ataxia, convulsions, coma.
- Breath often smells of eucalyptus oil.[7] This may be noticeable in the urine also.

Usual Fatal Dose
5 to 10 ml can cause serious toxicity.

Treatment
- Stomach wash.
- Supportive measures.

Forensic Significance
Most cases of poisoning are accidental, usually arising out of inadvertent overdose of the oil which is a popular household remedy for various ailments. Occasionally the oil has been administered in mistake for something else. Death is rare even in serious poisoning.

Azadirachta indica
Common Name
Neem

Characteristics
It is a tree that grows well all over India, and is revered for its 'medicinal properties'. The seeds yield a yellowish oil (**margosa oil**) which has a disagreeable odour and bitter taste.

Active Principle
The oil contains *linoleic, oleic, palmitic*, and *stearic acids*. Unrefined samples contain substantial quantities of *aflatoxins* which are injurious to the liver.

Uses
Margosa oil is used in the treatment of
- Skin diseases.
- Coughs and colds.
- Helminthiasis.

Clinical Features [8]
Ingestion of large quantities of margosa oil can cause serious hepatotoxicity characterised by vomiting, drowsiness, encephalopathy, and metabolic acidosis.

Treatment
- Rehydration.
- Treatment of cerebral oedema.
- Correction of metabolic acidosis.
- Supportive measures.

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Box 5.1 The Georgi Markov Case (1978)

Georgi Markov, a 49 year-old broadcaster with the BBC, was a Bulgarian who had defected to Britain. One day he was waiting near the Waterloo Bridge for his daily bus ride to the office, when he felt a sharp prick on the back of his right thigh. On turning around, he saw a man drop his umbrella. Apparently he had accidentally jabbed Markov with the tip of his umbrella. The man apologised and quickly walked away some distance before he hailed a taxicab. Markov got the impression he was a foreigner since he appeared to have had some difficulty communicating with the taxi driver before he boarded the vehicle.

At work on that day Markov did not feel very well. He left for home after his shift ended at 10.30 pm. By now he was experiencing gastrointestinal distress and had a high fever. He was taken to hospital where he recounted the incident at the bus stop, to the attending doctor. After investigations, a diagnosis of septicaemia was made. In spite of aggressive treatment, Markov died 3 days later. Since it was not clear as to what exactly was the cause of death, a medicolegal autopsy was carried out. Particular attention was paid to the dissection of right thigh where Markov had complained of being jabbed by the unknown assailant. Careful probing revealed a tiny metal pellet about 1.5 mm in diameter, which turned out to be an alloy of platinum and iridium. Two small holes had been bored into its casing. The pellet was subjected to chemical analysis and though conclusive evidence could not be obtained, the results suggested that the pellet had contained ricin. At the inquest, the Coroner returned a verdict of death due to ricin by unlawful administration. However, in spite of intensive police investigation, the culprit was never caught. It was most probably a case of political assassination.

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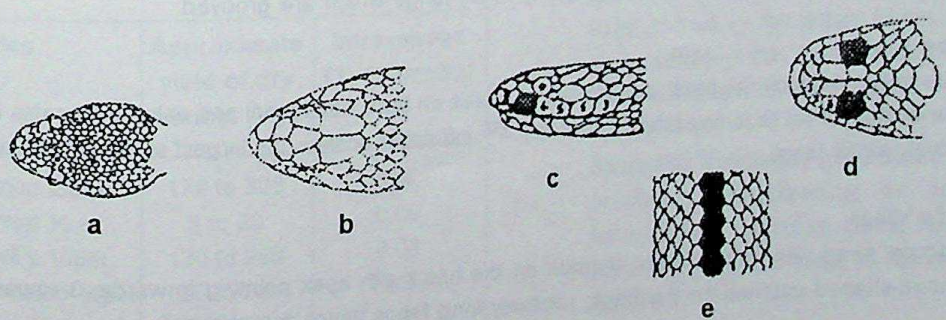
CHAPTER - 6

Bites and Stings

Envenomation can occur through the bites and stings of various creatures such as snakes, scorpions, spiders, bees, etc., leading to toxic manifestations, sometimes serious enough to cause death. The more common of these toxidromes will be discussed here.

SNAKE BITE

There are more than 2000 species of snakes in the world, and about 216 species in India of which 52 are venomous. Every year about 2 lakh individuals are bitten of whom an estimated 15000 subsequently die. However it is important to note that the majority of snakes are non-venomous, and their bites are not of much consequence. Doctors are advised to be familiar with the essential differences between venomous and non-venomous snakes so that needless administration of potentially harmful antivenom can be avoided. **Table 6.1** outlines major differences, while **Fig 6.1** illustrates some of them. In addition, some venomous snakes have very characteristic (even unique) features, and these have been described in **Table 6.2**. The commonest snakes encountered in India include Common Cobra, Common Krait, Russell's Viper, and Saw-scaled Viper.[1] Pit Vipers and Sea snakes are also encountered.



(a) Triangular head with small scales (b) Oval head with large scales (c) Third supralabial shield is the largest of the supralabials (d) Fourth infralabial shield is the largest of the infralabials (e) Central row of hexagonal vertebral scales.

Fig. 6.1 General features of poisonous snake

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Table 6.1 Differences between venomous and non-venomous snakes

Venomous Snake	Non-Venomous Snake
Usually stout, dull coloured, with abruptly tapering tail.	Usually slender, brightly coloured, with gradually tapering tail.
Tail may be rounded or flattened.	Tail always rounded.
Belly scales are broad.	Belly scales are small (do not extend across the entire width).
Head is usually triangular in shape.	Head is usually rounded or oval.
Head scales are usually small.	Head scales are usually large (shields).
At least one pair of teeth in the upper jaw are modified to form fangs which may be grooved or channelised.	All teeth are uniformly small in size, and are neither grooved nor channelised.
Saliva (venom) contains toxic peptides and enzymes.	Saliva is non-toxic.

Table 6.2 Unique features of some venomous snakes

Common Cobra

Large scales on head; presence of a hood with a spectacle or monocle mark; 3rd supralabial shield touches eye and nose shield; relatively small fangs which are grooved.

Common Krait

Bands or half rings on the back; central row of scales on back hexagonal and enlarged; scales on under-surface of tail (sub-caudates) undivided; 4th infralabial scale is the largest of the infralabials; relatively small fangs.

Russell's Viper

Triangular head with small scales; V mark on the head with apex pointing forwards; 3 rows of diamond-shaped patches on the back; relatively long fangs which are channelised.

Saw-scaled Viper

Triangular head with small scales; white arrow mark on the head; wavy line on each flank; scales of body serrated; relatively long fangs which are channelised.

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Snake Venom[2]

Source

It is the toxic saliva secreted by modified parotid salivary gland of a venomous snake.

Physical Appearance

Clear, amber-coloured fluid when fresh.

Constituents

1. *Toxins*: low molecular weight peptides & proteins.
2. *Enzymes*: Proteinases, hydrolases, transaminase, hyaluronidase, cholinesterase, phospholipase, ATPase, ribonuclease, deoxyribonuclease.
3. *Miscellaneous*: neurotoxin, cardiotoxin, haemolysin, haemorrhagins.

Nature

Conventionally, venom is classified as follows:

- *Neurotoxic* – elapid snakes, i.e., cobra, krait.
- *Haemotoxic* – viperid snakes, i.e., vipers.
- *Myotoxic* – hydrophid snakes, i.e., sea snakes.

The venom of krait and Russell's viper is much more toxic than that of cobra (**Table 6.3**).

Table 6.3 Venoms of Some Indian Snakes

Species	Approximate yield of dry venom (mg)	Intravenous LD 50 (mg/kg)
Common Cobra	170 to 325	0.4
Common Krait	8 to 20	0.09
Russell's Viper	130 to 250	0.08
Saw-scaled Viper	20 to 35	2.3
Pit Viper	40 to 60	6.2

Adapted from **Pillay VV.** *Influence of fang structure and venom composition on the symptomatology of snake bite.* J Forensic Med Toxicol **1996**; 13 : 18-20.

Symptomatology of Non-Venomous Snake Bite

The universal fear associated with snake bite induces a state of shock in many of these patients, characterised by syncope, hypotension, sweating, feeble pulse, and rapid, shallow breathing. The bite site may demonstrate multiple teeth impressions. Usually there is a lack of significant local pain or swelling.

Adequate reassurance and symptomatic treatment measures lead to full recovery.

Symptomatology of Venomous Snake Bite

Elapid bite

- **Local Features:** indistinct fang marks; burning pain; swelling and discolouration; sero-sanguinous discharge.

Systemic Features:

- *Pre-paralytic stage* - vomiting, headache, loss of consciousness.
- *Paralytic stage* - ptosis, ophthalmoplegia, drowsiness, dysarthria, dysphagia, convulsions, bulbar paralysis, respiratory failure.

Viperid bite

- **Local Features:** rapid swelling with discolouration and blister formation which may extend to the entire bitten limb and even involve the trunk; frank bleeding from the bite-site; severe pain.
- **Systemic Features:** generalised bleeding tendency manifesting as epistaxis, haemoptysis, bleeding gums, haematuria, melaena, purpuric spots in the skin; renal failure.

Hydrophid bite

- **Local Features:** minimal swelling and pain.
- **Systemic Features:** myalgia, muscle stiffness, myoglobinria, renal tubular necrosis.

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Treatment of Venomous Snake Bite

"Carefully identify the punctures made by the fangs.....excise the part with a sharp penknife.....round each puncture, deeply almost down to the bone....encourage bleeding.....then apply to the bottom of the wound a red-hot iron....." **Ewart J,** MD, MRCP. **(1878).**

We have come a long way since 1878 !

First aid measures[3]

- Reassurance.
- Keep the patient warm, and at rest.
- Immobilize bitten limb.
- Non-sedating, non-salicylate analgesic – paracetamol is best.
- Tourniquet – may be applied lightly, proximal to bite-site to prevent lymphatic spread of venom. It should be capable of admitting a finger under it. Distal pulse should be maintained. Once applied, it should not be loosened until antivenom therapy is begun, or it is confirmed to be a non-venomous bite. A modification of the tourniquet is the *Sutherland wrap*, i.e., application of a broad, firm, constrictive (crepe) bandage over the bitten area and also including the entire limb, with the limb placed in a splint.[4]
- Incision and suction is generally not advised.
- Cryotherapy (application of ice) is contraindicated.

Hospital measures

- Observe every case of alleged snake-bite for at least 24 hours before discharging.[5]
- The following must be monitored :
- Pulse rate, respiratory rate, blood pressure, WBC count, every hour.
- Blood urea, creatinine.
- Urine output.
- Vomiting, diarrhoea, abnormal bleeds.
- Extent of local swelling and necrosis.
- ECG, blood gas analysis.

Antivenin therapy -

Indications

Do not administer antivenin as a routine measure. It is associated with serious risk of allergic reactions. It should be given only when features of envenomation are present –

- Incoagulable blood.
- Spontaneous systemic bleeding.
- Hypotension.
- Neurotoxic or myotoxic features.
- Depressed consciousness.
- Expanding local swelling.
- Tender regional lymph nodes.

Nature: In India, only polyvalent antivenin is available, which is effective against the following snakes: common cobra, common krait, Russell's viper, and saw-scaled viper.

Sources:

- Haffkine Institute, Mumbai.
- King's Institute, Chennai
- Central Research Institute, Kasauli (Himachal Pradesh).
- Serum Institue, Pune.

Mode of administration : First perform a skin test as follows – inject intradermally 0.02 to 0.03 ml of a 1:10 dilution of antivenin in 0.9% saline. Positive test is indicated by development of urticarial wheal with erythema within 15 minutes. In such cases, desensitisation can be attempted by subcutaneous injection of 0.1, 0.2, and 0.5 ml of a 1:100 dilution of antivenin at 15 minute intervals. Subsequently a 1:10 dilution is given in the same manner, followed by undiluted antivenin. If no severe reaction occurs, the usual dose is given intravenously.

In non-allergic individuals, antivenin is always administered intravenously.

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Procedure: Dissolve the antivenin in distilled water or normal saline and administer the appropriate dose as an infusion in 500 ml of saline at 15 to 20 drops per minute. The rate can be progressively increased so that the infusion is completed in 1 to 2 hours.

Dose [6]:

Saw-scaled viper – 1 to 2 ampoules.
Sea snake – 3 to 4 ampoules.
Russell's viper – 8 to 10 ampoules.
Common cobra and krait – 5 to 20 ampoules.

The dose is same for children. Antivenin is not contraindicated in pregnancy.

Adverse reactions: Anaphylaxis. Always keep adrenaline loaded and ready.

Other measures

- Clean bite-site with povidone-iodine solution. Do not apply dressings.
- Leave blisters alone.
- If there is local necrosis, excise slough, and apply saline dressings.
- Tetanus toxoid.
- Prophylactic antibiotic (not essential).
- Paracetamol for pain.
- Rehydration and nutrition.

Special measures

- Severe anomalous bleeding may require administration of blood or fresh frozen plasma.
- If there are features of neurotoxicity (elapid bites), administer neostigmine 0.25 to 0.5 mg i.v, half hourly. Precede every injection with atropine 0.6 mg, i.v.
- Oxygen, assisted ventilation, etc., in case of respiratory failure.
- Treatment of renal failure along conventional lines.

Autopsy Features

- Signs of envenomation may not be readily apparent in every snake-bite fatality.
- Make a careful head-to-toe search with a hand lens for fang marks.
- Chemical analysis is usually unsuccessful in confirming a snake-bite. However, the skin around the bite site may be incised and submitted for laboratory testing, where-in an extract of the sample is injected into a frog and evidence of toxicity observed.[7]
- In unequivocal cases there may be evidence of fang marks, local swelling, blisters, bleeding into tissues, pulmonary oedema, froth in airways, cerebral oedema, etc., depending on the type of snake.

Forensic Significance

- Almost all the cases are accidental in nature.
- Homicides have been occasionally reported – e.g., casting an agitated snake on an unsuspecting or sleeping victim.
- Queen Cleopatra is believed to have committed suicide by having a venomous snake (an asp) bite her.

SCORPION STING

Scorpions belong to Class Arachnida (comprising eight-legged creatures), and are quite commonly encountered in India. Of the nearly 100 Indian species, the commonest are *Mesobuthus tamulus* (red scorpion), and *Palamneus swammerdami* (black scorpion). The former is more venomous. Venom which is a mixture of neurotoxic and cardiotoxic constituents, is contained in the stinger at the end of the tail.

Clinical Features

- Local burning pain, swelling, paraesthesias.
- Sweating, salivation, vomiting, abdominal cramps.
- Mydriasis.

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- Hypotension followed by hypertension, bradycardia followed by tachycardia, cardiac arrhythmias, myocarditis.
- Convulsions.
- Pulmonary oedema.
- Oliguria.

Treatment

- Immobilise the stung limb.
- Local ice application
- I.v fluids.
- Oxygen.
- Treatment of hypo-/hypertension.
- Diazepam for convulsions.
- Metoclopramide for vomiting.
- Furosemide for pulmonary oedema.

BEE & WASP STING

Bees and wasps belong to Class Insecta and Order Hymenoptera. Bees usually only sting once, while wasps are capable of repeated stings. However, retained stingers are common in the case of the former.

Clinical Features

- *Local reaction*: pain, swelling, oedema.
- *Anaphylactic reaction*: occurs in a previously sensitised individual. Features include facial flushing, urticaria, dry cough, dyspnoea, wheezing, stridor, abdominal cramps, vomiting, diarrhoea, shock.
- *Toxic reaction*: headache, fever, myalgia, vomiting, convulsions, syncope. Renal failure occurs occasionally.

Treatment

- Local cold compresses.
- Scraping away of retained stinger with scalpel blade (in bee sting).
- Antihistamines: diphenhydramine (50 mg, 6th hourly), or chlorpheniramine (4 mg 6th hourly).
- Adrenaline and oxygen for anaphylaxis.
- Salbutamol or aminophylline for bronchospasm.
- Analgesics for pain.

CANTHARIDES

A special type of insect-related poisoning occurs not due to a bite or sting by the creature, but due to eating it ! The insect in question is *Cantharis vesicatoria* (blister beetle). The killed and dried beetle is powdered (cantharides), and consumed in the belief that it is an aphrodisiac. This fallacious belief has its roots in the urinary tract irritation induced by the active principle cantharidin, resulting in exquisitely painful and frequent micturition, and priapism. Complications include haematuria, anuria, renal failure, vomiting, bloody diarrhoea, severe abdominal pain, convulsions, and delirium.

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CHAPTER - 7

Somniferous Poisons

The term *somniferous* means "sleep inducing", which is exactly what *narcosis* means. Hence a somniferous poison can also be referred to as a narcotic. The quintessential narcotic is **opium**. The following discussion revolves around this drug, as well some of its numerous derivatives.

Opium

Opium refers to the dried extract of the poppy plant (*Papaver somniferum*) which belongs to the family Papaveraceae and is a herb growing upto 1 metre in height. The leaves are oblong, irregularly toothed, and slightly lobed. Flowers are large and may be bluish white, purple, or white in colour. Each plant bears 5 to 8 capsules which are incised in their unripe state to extract a milky fluid that on drying yields opium, (Fig 7.1).

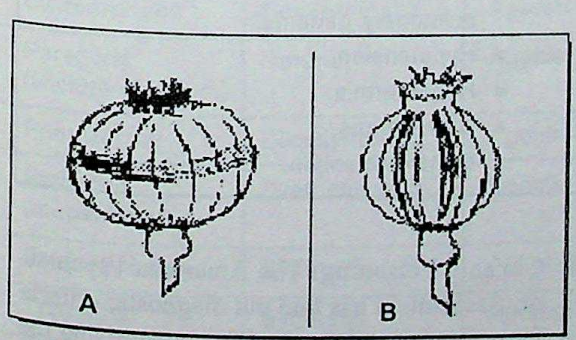

Fig. 7.1 Poppy capsule with (a) Turkish and (b) Indian incision

Papaver somniferum grows well in India, but its cultivation is banned except on license obtained from the central government, for growing the plant strictly for the pharmaceutical industry. Such a license is issued only for the states of Rajasthan, Uttar Pradesh, and Maharashtra. India produces 70 to 80% of opium that is used worldwide by pharmaceutical companies to manufacture several vital drugs including morphine, codeine, and pethidine. Unfortunately, a significant quantity of opium is funnelled clandestinely into a global smuggling racket which feeds the illicit drug trade flourishing in Western countries.

Physical Appearance

Crude opium is a dark brown or grey irregular mass with a characteristic odour and bitter taste.

Active Principles

Opium contains a number of alkaloids which fall into two groups -

1. *Phenanthrene group* : morphine, codeine, and thebaine.
2. *Benzylisoquinoline group* : papaverine and noscapine (narcotine).

Apart from these natural derivatives there are several other drugs used in pharmacotherapeutics which are either semisynthetic or synthetic analogues (Table 7.1). When referring to these de-

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rivatives, there has always been confusion in terminology. While strictly speaking, the term "opiate" refers to natural and semisynthetic derivatives, and the term "opioid" is a more inclusive term applying to all agonists and antagonists with morphine-like activity, for the sake of convenience (and to avoid confusion), the former term will be used in this book as an umbrella-expression to cover all derivatives of opium – natural, semisynthetic, and synthetic.

Mode of Action

There are 3 major classes of opiate receptors to which different opiates bind with differing affinity.

- The m (mu) receptor - Most of the clinically used opiates are relatively selective for m receptors reflecting their similarity to morphine. m_1 subtype is responsible for supraspinal analgesia, peripheral analgesia, and euphoria, while m_2 subtype is responsible for spinal analgesia, respiratory depression, physical dependence, gastrointestinal dysmotility and miosis.[1]
- The k (kappa) receptor - k_1 is responsible for spinal analgesia and miosis, k_2 is associated with psychotomimesis and dysphoria, while k_3 is responsible for supraspinal analgesia.[2]
- The d (delta) receptor - is said to be important in spinal and supraspinal analgesia.[3]

Uses

Opiates are extensively used in pharmacotherapeutics (**Refer Table 7.1**).

Toxicokinetics

In general most opiates are readily absorbed from the g.i tract, and also can be administered by subcutaneous, intramuscular or intravenous injection. However the effect of an oral dose is usually much less than that obtained on parenteral administration due to significant first-pass metabolism in the liver. For example, the bioavailability of oral morphine is only 25%. Extent of protein-binding is variable depending on the exact nature of the opiate, and varies from 7% for codeine to 96% for buprenorphine. Morphine displays protein-binding to the extent of 34%. The major metabolic pathway of morphine is conjugation with glucuronic acid to produce morphine-6-glucuronide which is also pharmacologically active. Excretion occurs in the urine as morphine-3-glucuronide. Duration of action of opiates varies from 2 hours (pentazocine, pethidine) to 4 hours (morphine, codeine, heroin), to 8 hours (methadone, buprenorphine). Fentanyl, alfentanil, and surfentanil are ultra-shortacting (few minutes to 1 hour).

Toxic Effects

I. **Acute Poisoning**: The triad of coma, pinpoint pupils, and respiratory depression is said to be almost pathognomonic for opiate poisoning.[4] Such cases may be due to therapeutic overdose, accidental overdose (in addicts), or deliberate overdose (suicidal). The important clinical manifestations are summarised in **Table 7.2**

Table 7.2 Acute Opiate Poisoning

- Coma
- Pinpoint pupils (may be dilated if hypoxia is severe).
- Bradypnoea, cyanosis, non-cardiogenic pulmonary oedema.
- Hypotension.
- Hypothermia.
- Convulsions.
- Urinary retention.

II. **Chronic Poisoning**: The American Psychiatric Association has laid out diagnostic criteria for opiate dependence and for categorising the severity of such dependence.

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Table 7.1 Opium Derivatives

Derivative	Nature	Classification	Use
Buprenorphine	Semisynthetic	Partial agonist	Analgesic, pre-anaesthetic medication
Butorphanol	Semisynthetic	Agonist-antagonist	Analgesic
Codeine	Natural	Agonist	Antitussive
Dextromethorphan	Semisynthetic	—	Antitussive
Diphenoxylate	Synthetic	Agonist	Antidiarrhoeal
Fentanyl	Synthetic	Agonist	Adjunct to anaesthesia
Heroin	Semisynthetic	Agonist	—
Hydrocodone	Semisynthetic	Agonist	—
Hydromorphone	Semisynthetic	Agonist	—
Levorphanol	Semisynthetic	Agonist	Analgesic
Loperamide	Synthetic	Agonist	Antidiarrhoeal
Methadone	Synthetic	Agonist	Analgesic, treatment of heroin abuse and opiate abstinence syndrome
Morphine	Natural	Agonist	Analgesic
Nalbuphine	Semisynthetic	Agonist-antagonist	Analgesic
Nalmefene	Semisynthetic	Antagonist	Treatment of opiate poisoning
Naloxone	Semisynthetic	Antagonist	Treatment of opiate poisoning
Naltrexone	Semisynthetic	Antagonist	Treatment of opiate poisoning and alcoholism
Oxycodone	Semisynthetic	Agonist	Analgesic
Oxymorphone	Semisynthetic	Agonist	—
Paregoric (tincture of opium)	Natural	Agonist	Analgesic
Pentazocine	Semisynthetic	Agonist-antagonist	Analgesic
Pethidine (meperidine)	Synthetic	Agonist	Analgesic
Propoxyphene	Synthetic	Agonist	Analgesic
Tramadol	Synthetic	Agonist	Analgesic

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The following are some useful pointers which indicate opiate addiction[5]:

- Unusual mood swings, periods of depression alternating with euphoria.
- Withdrawal from family, friends, and social activities.
- Frequent domestic strife.
- Long hours of unexplained absence from home
- Unexplained overspending.
- Frequent conflicts with law (e.g., driving offences).
- Dwindling sexual drive.
- Pills, syringes, etc., lying around the house.
- Bloody swabs or tissues lying around the home or in the working place.
- Periodic disappearances into a locked room (bathroom, bedroom, etc.).
- Pinpoint pupils.
- Weight loss, pallor.
- Chronic constipation.
- Periodic withdrawal manifestations – sweating, tremors.

In addition, an addict may have dermal scars (from intravenous abuse), and suffer from amnesia, confusion, and occasional hallucinations.

Abrupt cessation of opiate intake can cause a withdrawal reaction (*cold turkey*). Common manifestations are mentioned in (**Table 7.3**).

Usual Fatal Dose

The usual fatal doses (as well as usual therapeutic doses) of common opiates are listed in **Table 7.4**.

Table 7.4 Usual Fatal Dose & Usual Therapeutic Dose For Common Opiates

Opiate	Usual Fatal Dose	Usual Therapeutic Dose
Morphine	200 mg	10 to 15 mg
Codeine	800 mg	10 to 60 mg
Heroin	50 mg	—
Crude Opium	500 mg	—
Pethidine	1 gm	50 to 150 mg
Methadone	100 mg	5 to 10 mg
Pentazocine	300 mg	30 to 60 mg
Propoxyphene	1 gm	100 to 150 mg
Diphenoxylate	200 mg	10 to 20 mg

Diagnosis

1. Needle marks, dermal scars (suggestive of addiction).

Table 7.3 Opiate Withdrawal

Anticipatory (3 to 4 hours)	Early (8 to 10 hours)	Fully Developed (1 to 3 days)	Protracted Abstinence (upto 6 months)
Anxiety, craving, drug-seeking behaviour	Restlessness, yawning, nausea, sweating, rhinorrhoea, lacrimation, mydriasis, stomach cramps, drug-seeking behaviour	Tremor, piloerection, vomiting, diarrhoea, muscle spasm, hypertension, tachycardia, fever, chills, impulse driven drug-seeking behaviour	Hypotension, bradycardia insomnia, anorexia, stimulus driven opiate craving

** Times mentioned in the table refer to heroin.*

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2. Evidence of hypoglycaemia, hypoxia, and hypothermia.
3. Most opiates can be detected in urine or blood by RIA, GC, GC-MS, or HPLC.
4. Empirical administration of naloxone, (can precipitate reaction in addicts).

Treatment

I. **Acute Poisoning***:
   1. Supportive measures –
      - Maintenance of patent airway.
      - Endotracheal intubation, assisted ventilation.
   2. Naloxone is the antidote of choice for opiate poisoning, (**Box 7.1**).
   3. The use of physostigmine salicylate (0.04 mg/kg i.v) has been suggested for reversing respiratory depression if the regular opiate antidotes are not available, since it increases the acetylcholine content of the reticular formation of the brainstem which is suppressed by opiates.[6]
   4. The use of drugs such as levorphanol and amiphenazole is no more recommended today.
   5. Convulsions may be treated with benzodiazepines in the usual manner, though this is frequently not necessary if naloxone is available.

II. **Chronic Poisoning**:
   1. Gradual withdrawal of the opiate.
   2. Substitution therapy with methadone begun at 30 to 40 mg/day and then gradually tapered off.
   3. A beta adrenergic blocker like propranolol (80 mg) is said to be quite effective in relieving the anxiety and craving associated with opiate addiction, but has no effect on physical symptoms. Alternatively, drugs such as clonidine can be used.[7] Buprenorphine or naltrexone can also be used.
   4. Antispamodics for abdominal cramps associated with vomiting and diarrhoea.
   5. Tranquillisers or bed time sedation if necessary.
   6. Psychiatric counselling.

Autopsy Features [8]

- Injection marks, dermal abscesses, scarring. Look for injection marks in the antecubital fossae, forearms, back of the hands, neck, groin, and ankles.
- Tattooing, (a common feature of the drug subculture).
- Emaciation, unkempt appearance.
- Gross pulmonary oedema with froth exuding out of mouth and nostrils, especially in sudden heroin-related death.
- Cerebral oedema.
- Congestion of liver with enlargement of hepatic lymph nodes. Chemical analysis of lymph nodes may reveal presence of morphine.**

Samples of blood, urine, brain, liver, and bile must always be preserved for chemical analysis.

It is important to remember that infections such as serum hepatitis and AIDS are common among intravenous drug abusers, and hence autopsies conducted in drug-related deaths must be done cautiously with necessary precautions.

Forensic Significance

Opiates are among the commonest of the drugs abused today in India. Heroin (brown sugar) is popular among all classes of addicts in metropolitan cities such as Mumbai, Delhi, Bangalore, etc. Opiates used for therapeutic purposes, for e.g.,

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* *With particular reference to **morphine**, the archetypal opiate.*

** *Even if heroin was being abused by the victim, it is metabolised into morphine in the body.*

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morphine, pethidine, and pentazocine are more commonly abused by medical and paramedical personnel. Codeine which is easily available over the counter in the form of antitussive preparations is being increasingly abused, especially by college-going youth.

Accidental deaths are not infrequent from overdose, particularly among intravenous abusers of heroin ("death on the needle"). Suicidal deaths are also reported from time to time since opiates are reputed to cause a painless death. Homicides are rare, but a few cases have been documented in medical literature.

Box 7.1
Antidotes for Opiates

The first opiate antagonist to be introduced into practice was N-allylnorcodeine in 1915. Nalorphine was developed in 1954, naloxone in 1960, and naltrexone in 1963. The most recent entrant is nalmefene. Nalorphine and levallorphan (a related antagonist) are no more used today since both are weak competitive antagonists with undesirable k-receptor agonist properties.

Naloxone, naltrexone, and nalmefene (all derivatives of oxymorphone) are pure competitive antagonists at m, k, and d receptors. They are recommended today as approved antidotes in opiate poisoning.

Naloxone
It is effective against all opiates including pentazocine, but is not very effective against buprenorphine.[1] Dramatic reversal of the following features is achieved : miosis, respiratory depression, hypotension, and coma.

Dose: The usual initial dose is 1.2 mg for an adult and 0.4 mg for a child. The best route of administration is i.v. Since the effect of a single bolus dose of naloxone is usually short-lived, repeated doses are required.

There are reports suggesting the utility of naloxone in overdoses of captopril and valproic acid.[2,3] Naloxone may also be useful in acute alcohol poisoning.[4]

Indian Brand Name : Narcotan (Troikaa), 2 ml ampoules of 20 mcg/ml, and 1 ml ampoules of 400 mcg/ml.

Naltrexone: Naltrexone is a long-acting opiate antagonist which can be administered orally. It is usually used for treating opiate addiction. However, it must not be given to an opiate-dependent patient who has not been detoxified.[5] A challenge dose of naloxone to confirm the lack of opiate dependence is recommended before beginning naltrexone.

Dose: 50 mg/day orally, which may have to be continued for several weeks or months.
Indian Brand Name : Naltima (Intas), 50 mg tabs.

Nalmefene: Nalmefene is a naltrexone derivative with pure opiate antagonistic effects, and has a longer duration of effect than naloxone in acute opiate poisoning.[6] It is usually given intravenously beginning with 0.1 mg, and if withdrawal reaction does not occur, 0.5 mg is administered, followed by 1 mg in 2 to 5 minutes (if necessary).

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ADDITIONAL FEATURES WITH REFERENCE TO INDIVIDUAL OPIATES (in alphabetical order)

Codeine (Methylmorphine)

Codeine is 60% as effective orally as parenterally, its oral efficacy being the result of less first-pass hepatic metabolism. It has very low affinity for opiate receptors and its analgesic effect is mainly due to conversion to morphine. However its antitussive action involves distinct receptors that bind codeine itself.

Today in some parts of the world, codeine is being used for illicit synthesis of morphine and heroin involving a simple demethylation procedure. The resulting product is referred to as "home bake." [9] In parts of the USA, oral combinations of glutethimide and codeine ("sets") are being abused. Deaths have been reported among such "sets" abusers.[10]

The lethal blood level of codeine is said to be approximately 0.5 mg/100 ml.[11]

Fentanyl

Fentanyl is a synthetic opiate related to the phenylpiperidines and is primarily an m agonist being 80 times as potent as morphine (as an analgesic). Sufentanil and alfentanil which are congeners of fentanyl are even more powerful. Fentanyl is used as an anaesthetic either alone or in combination with droperidol. Congeners of fentanyl are also employed as anaesthetics.

Today fentanyl and its analogues are abused extensively in some western countries since they induce a much stronger euphoria than heroin and other opiates.[12] These drugs may be sniffed, smoked, injected, rubbed on the buccal mucosa, or gargled. Some fentanyl derivatives such as a-methylfentanyl (China white), 3-methylfentanyl, and fluorofentanyl are referred to as "designer drugs"* and are extremely popular abroad as recreational drugs.[13]

Heroin (Diacetylmorphine)

Heroin has no role in therapeutics and yet is the most widely abused of all the opiates. It was introduced in 1898 and was touted as a heroic cure for morphine addiction, (hence the name). A greater irony would be hard to find !

** Designer drugs are substances modified from legitimate therapeutic drugs for recreational use.*

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Heroin was extremely popular till the late 1970s and early 1980s, but thereafter a decline was noticed probably because of the fear of AIDS associated with its (intravenous) abuse. However it is now making a major comeback in a cheaper and much purer form that can be smoked or snorted.

Because of its high solubility, heroin crosses the blood-brain barrier quickly and is de-acetylated to the active metabolites, 6-monoacetylmorphine and morphine. After the intense euphoria ("rush") which is akin to a sexual orgasm, lasting from one to several minutes, there is a period of sedation ("on the nod") which lasts for about an hour. The effects completely wear off in about 3 to 6 hours, so that a habitual user usually feels the need to inject himself 2 to 4 times a day. There is a tendency to constantly increase the daily dose on account of tolerance. Combination of heroin with other drugs is common, for e.g., with cocaine (*speedball*), amphetamines, alcohol, benzodiazepines, or cannabis. Combination with phencyclidine can cause death.[14] Street heroin (*brown sugar*) is often laced with additives and adulterants. Deliberate adulteration is often done by a pusher in order to increase the margin of profit. This is referred to as "cutting". Sometimes cutting is resorted to as a method of getting rid of a bad payer or a potential traitor by adding a lethal substance such as strychnine (*hot shot*).

Body Packer Syndrome – Although this is more commonly associated with smuggling of cocaine, it has also been reported in the case of other drugs, especially heroin. A "bodypacker" or "mule" is an individual who attempts to transport illicit drugs from one country to another by ingesting wrapped packages, or condoms, or balloons containing concentrated cocaine or heroin. After arrival at the destination, cathartics are self-administered and the packets are defaecated out. Sometimes rectal suppositories or disposable enemas are used. Although generally asymptomatic, in a few cases serious toxicity may result due to rupture of packets.[15] Diagnosis of an asymptomatic body packer can be accomplished with the help of an abdominal x-ray, ultrasound, or CT scan. The last two methods should be resorted to only if x-ray is unclear. In some cases, faecal examination over a period of a few days may be necessary.[16]

Pethidine (Meperidine)

Pethidine is a phenylpiperidine and is a primary m agonist producing effects which are similar to those of morphine. In general, 100 mg of pethidine given parenterally is approximately equal to 10 mg of morphine. It is only half as effective on oral administration. Contrary to popular misconception, pethidine usually produces miosis like all other opiates and not mydriasis. The main difference with morphine relates to CNS manifestations during toxicity. Pethidine sometimes produces CNS excitation characterised by tremor, muscle twitching, and convulsions, probably due to the action exerted by one of its metabolites – norpethidine (normeperidine).[17] In such an eventuality, there may be mydriasis.

Acute pethidine poisoning is treated with naloxone (opiate effects), diazepam or intravenous barbiturates (convulsions), and carbamazepine (hypomania).

Although pethidine abuse does exist, it is mostly restricted to those having access to the drug such as medical or paramedical professionals, since it is not widely available as an illicit drug. However an inadvertent catastrophe occurred in 1982 in the USA, when during attempts to illicitly synthesise a pethidine analogue MPPP (1-methyl-4-phenyl-4-propionoxy-piperidine), a variant was produced: MPTP (1-methyl-4-phenyl-1,2,3,6-tetrahydropyridine), which induced severe parkinsonian symptoms in those who used it. Due to the obdurate akinesia, these unfortunate individuals were labelled as "frozen addicts".[18] Anti – parkinsonian

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drugs did not produce much relief, and several of the affected victims had to undergo stereotactic implantation of foetal tissue into their basal ganglia.[19]

Poppy Seeds

Contrary to popular belief, poppy seeds which are used widely for culinary purposes do contain traces of morphine and codeine. Eating a large quantity of these seeds can lead to positive screening for both the alkaloids.[20]

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CHAPTER - 8

Inebriant Poisons

The term **inebriant** refers to any substance that intoxicates, i.e., induces mental confusion, lightheadedness, disorientation, and drowsiness. It naturally follows that the most important representatives are the **alcohols**. There are several other poisons and drugs which also fall under this category: **barbiturates, chloral hydrate, benzodiazepines, hydrocarbons, formaldehyde, paraldehyde,** and **anaesthetic drugs**. While many of the **pesticides** in common use are conventionally classified as inebriants, they will be discussed separately.

ALCOHOLS

Alcohols are actually (hydroxy) derivatives of (aliphatic) hydrocarbons. There are 3 categories of alcohols –

1. *Monohydroxy alcohols:* They have only one hydroxyl (OH) group. E.g., ethanol, methanol, isopropanol, etc.
2. *Dihydroxy alcohols:* They possess two hydroxyl groups and are referred to as glycols. E.g., ethylene glycol, propylene glycol, etc.
3. *Trihydroxy alcohols:* They are not really alcohols, but only derivatives. E.g., propane derivative glycerol or glycerine.

Ethanol

Synonyms

Ethyl alcohol, grain alcohol

Physical Appearance

Clear, colourless liquid with a faint fruity odour, and sweetish burning taste. It is both water soluble and lipid soluble.

Sources

Ethanol is produced by fermentation of a sugar (from cereal, vegetable, or fruit) with yeast. If cereal is used as raw material, it has to be malted first to convert the starch to maltose, since yeast cannot ferment starch. Strong alcoholic beverages are distilled after fermentation. The Arabs were responsible for introducing distilled spirits into the Western world in the Middle Ages. The Europeans were so taken up by the refined taste and effects of these spirits, particularly whisky, that they became convinced that alcohol was the long sought elixir of life.*

The ethanol content of various alcoholic beverages is expressed by volume percent or by proof, the latter being twice the percentage of alcohol by volume. *Proof spirit* refers to a standard mix-

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* *In fact, the term whisky means "water of life" in Gaelic ("usquebaugh" being the original term).*

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ture of alcohol and water of relative density 12/13 at 51° F, i.e., 49.28% of alcohol by weight (or 57.10% by volume). Proof strength of alcoholic beverages is expressed in degrees. **Table 8.1** lists the ethanol content of various alcoholic beverages.

Table 8.1 Ethanol content in alcoholic beverages

Beverage	Ethanol Content (percentage by volume)
Light Beer (Lager and Pilsener)	4 to 6
Heavy Beer (Ale and Stout)	6 to 8
Natural Wine (Claret and Cider)	10 to 15
Fortified Wine (Sherry and Port)	15 to 20
Whisky, Gin, and Brandy	40 to 45
Rum	50

Apart from ethanol however, these beverages also contain several congeners to varying extent e.g., low molecular weight alcohols such as methanol and butanol, as well as aldehydes, esters, phenols, tannins, and heavy metals (lead, cobalt, iron, etc). Vodka is the purest form and contains no congeners[1]. It is virtually odourless. White rum is also relatively pure.

Uses

1. *Beverage:* Popular alcoholic beverages include beer, wine, whisky, gin, brandy, rum, and vodka (**Table 8.2**). In addition there are several indigenous preparations peculiar to particular regions, e.g., arrack, toddy, and feni in India, tequila in Mexico, sake in Japan, eau de vie or fruit brandy in France, etc.
2. *Solvent* for after-shaves, colognes, mouthwashes, and perfumes. The alcohol content in these is variable (15 to 80 %).
3. *Medicinal* -

Table 8.2 World's favourite alcoholic beverages*

Alcoholic Beverage	Countries (where the beverage is most popular)
Beer	Australia, Canada, Denmark, England, New Zealand
Brandy	France, Germany, Holland
Gin	Holland
Rum	West Indies
Vodka	Poland, Russia
Whisky	Canada, Ireland, Japan, Scotland, USA,
Wine	France, Italy, Portugal, Spain, Switzerland

** India shows no clear-cut preferential attitudes, but wine is generally not very popular. Distilled spirit consumption is more common in the cold northern parts, while beer is relatively more popular in the South.*

a) Several antihistaminic, decongestant, multivitamin, and cough syrups contain varying percentage of alcohol (2 to 25 %).
b) Ethanol has been popular in the past as an antiseptic. **Surgical spirit** used even today is mostly ethanol with a small quantity of methanol (90 to 95 % and 10 to 5 % respectively), along with traces of castor oil and methyl salicylate.
c) Ethanol sponging is an effective remedy for hyperthermia.
d) Injection of dehydrated alcohol in close proximity of nerves or sympathetic ganglia is said to be effective for the relief of long lasting pain in conditions such as trigeminal neuralgia[2].
e) Small doses of ethanol are said to be effective in minimising the severity of common cold.*

"At the first inkling of a cold, hang your hat on the bed post, drink from a bottle of good whisky until two hats appear, then get into bed and stay there !" **Hamburger (1936).*

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4. *Antidote* for methanol and ethylene glycol.
5. *Preservative*: **Rectified spirit** (90 to 95 % ethanol) is used as a preservative for viscera, for chemical analysis.
6. Fuel.

Usual Fatal Dose

One pint (approximately 550 ml) or quart (two pints or approximately 1100 ml) of a strong distilled spirit such as whisky taken in a short span of time can be lethal.

The usual fatal dose corresponds to approximately 6 gm of ethanol / Kg body weight (adult); 3 gm / Kg (child)[3]

In terms of blood alcohol, a level in excess of 400 to 500mg/100 ml is usually considered to be lethal.

Toxicokinetics

Ethanol is rapidly absorbed from the stomach (20%) and small intestine (80%). Maximum or peak alcohol concentration in blood is reached in 30 to 90 minutes following the last drink. Many factors can delay absorption: undiluted ethanol (by provoking pylorospasm), presence of food, delayed gastric emptying due to any cause, and presence of congeners in alcohol.

More than 90% of ethanol ingested is metabolised in the body, and only 5 to 10% is excreted unchanged by the kidneys, lungs, and sweat. Excretion of ethanol by the lungs obeys **Henry's Law** : the ratio between the concentration of ethanol in the alveolar air and the blood is constant. This alveolar air / blood constant (1 : 2100) forms the basis for reliably estimating blood alcohol concentration by breath analysis.

Metabolism of alcohol is accomplished through 3 pathways in the liver [4]

- Alcohol dehydrogenase pathway (in the cell cytosol)
- Microsomal ethanol oxidising system (MEOS, located on the endoplasmic reticulum)
- Peroxidase-catalase system (in the hepatic peroxisomes)

The first is the main pathway, while the third is the least important. The acetaldehyde that is formed is converted to acetic acid by aldehyde dehydrogenase, which in turn is converted to acetylcoenzyme A and enters the Krebs (citric acid) cycle where it is metabolised to carbondioxide and water.

In adults, the average rate of ethanol metabolism is 100 to 125 mg/kg/hr in occasional drinkers, and upto 175 mg/kg/hr in habitual drinkers.[5] The blood alcohol level generally falls at a rate of 15 to 20 mg/100 ml/hr. This may be higher (upto 30 mg/100 ml/hr) in chronic alcoholics. [6]

Mode of Action

For long, it was postulated that ethanol depresses the CNS by dissolving in the cell's lipid membrane and causing disorganisation of the lipid matrix (membrane fluidisation). However this mechanism has been challenged by recent studies

Now there are two theories which are gaining popularity. According to one, ethanol acts by enhancing GABA-nergic function through interaction with $GABA_A$ receptors and associated chloride ion channels.[7] The second theory which appears to be more convincing has to do with N-methyl-d-aspartate (NMDA) ligand-gated, glutamate receptors. NMDA receptors mediate neurotoxicity by increasing permeability to calcium, and regulate neuronal long term potentiation. Studies demonstrate that in the acute form of ethanol use, NMDA receptor function is inhibited, while chronic ethanol use results in up-regulation of NMDA receptors.[8]

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Pharmacological effects of ethanol

CNS: Ethanol is a CNS depressant but produces some apparently stimulating effects initially because of depression of inhibitory control mechanisms in the brain. First, those mental processes which depend on training and previous experience are affected (memory, concentration, and insight). Later the person becomes expansive, garrulous and may demonstrate emotional lability with mood swings. There are acompanying sensory and motor disturbances. With severe intoxication there is general impairment of CNS function, and finally coma supervenes.

CVS: In moderate doses, ethanol produces tachycardia and vasodilation of cutaneous vessels with resultant warm and flushed skin. There have been numerous studies documenting the beneficial effects of alcohol on cardiac health, and these have been critically analysed in **Box 8.1**.

GIT: Ethanol normally stimulates salivary and gastric secretions, but if the concentration is too high (> 40%) they are inhibited, and the g.i. mucosa becomes congested and inflamed leading to erosive gastritis.

Genito-urinary: Ethanol induces diuresis by inhibition of antidiuretic hormone (ADH). There is a popular misconception (perpetuated in pulp fiction and films) that ethanol is an aphrodisiac. While it is true that there is often enhanced sexual inclination with (sometimes) aggressive behaviour, this is due to loss of inhibition and restraint rather than the result of sexual stimulation.

Clinical Features

A. **Acute Poisoning** (Intoxication, Inebriation): Initially, ethanol produces excitement which progresses to loss of restraint, behavioural changes, garrulousness, slurred speech, ataxia, unsteady gait, drowsiness, stupor, and finally coma (**Table 8.3**).

Through all the 7 stages of ethanol intoxication, a distinct odour is perceptible in the breath of the individual. It is however not the alcohol itself which imparts this odour but other nonalcoholic constituents that give a particular flavour depending on the type of beverage consumed (wine, beer, whisky, etc).

Ethanol is a mydriatic, but towards terminal stages (stages of stupor and coma), the pupils may become constricted, only to dilate once again at about the time of death. The **McEwan sign*** (a perennial favourite of Toxicology textbooks) is highly unreliable and must not be depended upon for diagnosis of alcoholic coma.

* *Pinching the skin of the face or neck, or light slapping, is supposed to dilate the constricted pupils of alcoholic coma momentarily.*

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Box 8.1
Ethanol and Cardiac Health

"The injurious effects of alcohol must not be construed as the result of the use of a bad thing ; it is actually the result of abuse of a good thing" – **Abraham Lincoln**

Recent attention in the medical press has focussed on the reported association between "moderate" alcohol consumption and decreased risk of sudden death, especially from cardiovascular causes.[1] This has met with angry angry reactions from anti-liquor campaigners culminating in an emotional and sometimes bitter controversy. The following discussion is an attempt at presenting and analysing the facts available at present in a critical, unbiased manner.

To begin with, it must be admitted indubitably that alcohol is a vasodilator. But this is not the reason for alcohol's beneficial effects on the heart since the vasodilation is mostly confined to cutaneous vessels producing the warm, flushed skin of acute intoxication. There is virtually no increase in coronary blood flow in human beings. In fact, in individuals with existing coronary artery disease, ethanol decreases the time period of exercise required to precipitate angina and to produce changes in the ECG which are indicative of myocardial ischaemia.[2]

However strangely, several studies in the recent past indicated a clear negative corelation between chronic ingestion of small amounts of ethanol and the incidence of coronary heart disease, beginning with the **Kaiser-Permanente** epidemiologic study done way back in **1974** which was at first received with scepticism by the scientific community.[3] But it marked the beginning of a barrage of studies all around the globe over the next two decades establishing the clear association between moderate alcohol consumption and reduced risk of coronary heart disease.[4,5] So, what could be the reason ? After much study and deliberation investigators have now come to the conclusion that the protective effect of ethanol on the heart is because it increases the concentration of high density lipoproteins (HDL) in the plasma, while at the same time decreasing that of low density lipoproteins (LDL). It is well known that the lower the concentration of HDL in the blood the greater is the risk for developing CHD. The reverse is true for LDL. Today several studies have established a convincing relationship between alcohol intake and the level of protective HDL, including both its HDL2 and HDL3 subfractions.[6]

It is now also clear that apart from its beneficial effect on cholesterol levels, alcohol has favourable effects on haemostatic factors such as plasma fibrinogen, fibrinolytic activity, and platelet adhesiveness. Even forensic autopsies have pointed towards a positive association between moderate drinking and cardiac health.[7]

Having come to this conclusion, it must be emphaised that the purported beneficial effects rapidly evaporate as consumption is increased beyond desirable levels, and in fact the deleterious effects would then be much more than in those who don't drink at all.[8] So the question arises: what is "moderate" alcohol consumption ? Unfortunately the definition of moderation varies from one study to another, but the balance of evidence is that the daily consumption must not exceed 1 to 2 drinks per day. A standard drink is roughly the equivalent of 45 ml of distilled spirits (15.1 gm alcohol) or 150 ml of wine (10.8 gm alcohol), or 350 ml of beer (13.2 gm alcohol).[9]

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contd.

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In the final analysis, while physicians may use their discretion in advising patients about the beneficial effects of moderate ethanol consumption, it must be emphasised that safe drinking is not a panacea for sound cardiac health. The benefits documented thus far need further study and irrefutable confirmation. A disturbing new information is that even moderate ethanol intake can increase the risk of certain types of cancer.[10] Also, while moderate drinking may be beneficial to cardiac health in middle-aged people, its influence on young individuals is not very clear. In fact, a recent study indicates deleterious effects especially in the form of increased tendency to cerebral stroke and enhanced overall mortality among young men who consumed alcohol regularly.[11] In any case, advice advocating the use of moderate ethanol consumption is outright contraindicated in the following cases: alcoholic patient (one who has no control over his drinking), existing disease which can be aggravated by alcohol, patients on medication which can interact with alcohol, mental disease, and old age.[12]

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Table 8.3 Acute Alcohol Poisoning

Blood Alcohol Concentration (mg/100ml)	Stage of Intoxication	Clinical Features
0 – 50	Sobriety	Near normal behaviour
50 – 100	Euphoria	Feeling of well being, sociability, talkativeness, increased self confidence, decreased inhibitions, fine movements affected
100 – 150	Excitement	Emotional instability, impairment of memory and comprehension, increased reaction time, mild ataxia
150 – 200	Confusion	Disorientation, confusion, vertigo, diplopia, ataxia, slurred speech, staggering gait
200 – 300	Stupor	General inertia, diminished response to stimuli, inability to stand or walk, vomiting
300 – 500	Coma	Unconsciousness, abolished reflexes, subnormal temperature, incontinence of urine and faeces, respiratory compromise
> 500	Death	Death due to respiratory failure

The 7 stages can be remembered as a series of D's: Decent, Delighted, Delirious, Dazed, Dejected, Dead drunk, Dead.

Medicolegally, stages 3 and 4 of alcoholic intoxication (stages of excitement and confusion) are the most important, since most of the offences associated with drinking are committed during these two stages. It is pertinent to mention that stage 7 (death) is extremely rare in pure ethanol ingestion. In most cases there is recovery after prolonged sleep, with some residual after effects (**hangover**), consisting of headache, irritability, lethargy, nausea, and abdominal discomfort. While the last mentioned is mainly due to gastritis produced by ethanol, the other symptoms are actually the result of congeners and impurities present in alcoholic beverages which cause mild cerebral oedema. Part of the hangover may also be the result of hypoglycaemia induced by ethanol.

Alcoholic intoxication (through all the stages) can mimic several conditions which can lead to errors in diagnosis. **Table 8.4** lists the differential diagnosis for acute ethanol intoxication.

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Table 8.4 Differential diagnosis of alcoholic intoxication

Pre-Coma	Coma
Barbiturate ingestion (and other similar drugs)	Hypoglycaemic or Diabetic coma
Carbon monoxide poisoning	Coma due to hypnotics, sedatives, and other drugs
Hypoglycaemia	
Head injury (post concussional state)	Cerebrovascular accident
Psychotic states Disseminated sclerosis (and other similar neurological afflictions)	

Diagnosis

1. *Bedside test:* Place 1 ml of unknown solution plus 1 ml of acetic acid and 1 drop of H_2SO_4 in a test tube and heat gently for 1 minute. A characteristic, strong fruity odour (due to ethyl acetate) is positive for ethanol.

2. *Blood alcohol level:* The blood alcohol concentration (BAC) can be estimated by microdiffusion technique or by an electrochemical method. The latter is a rapid quantitative test which can be done in the emergency (casualty) department. BAC estimated by immunoassay or gas chromatography is the commonest method employed by laboratories in India. Although accurate, the results of these tests are often delayed several hours and are not readily appropriate in the clinical scenario.

Estimation of BAC indirectly by analysing the breath alcohol with the help of breathalysers is discussed on page no 375.

B. **Chronic Poisoning** (Alcoholism, Ethanolism): Alcoholism is a condition in an individual who consumes large amounts of alcohol over a long period of time. It is characterised by
   - a pathological desire for alcohol intake
   - black-outs during intoxication
   - withdrawal symptoms on ceasing alcohol intake

There are indications that alcoholism instead of representing a defect in moral character as traditionally believed, may actually be a genetically influenced, multifactorial disorder. Environmental and cultural influences are also very important. Alcohol related problems are rare among Jews and Muslims (in Islamic countries), while they are common in countries which have large alcohol-producing industries, for e.g., France, Italy, Portugal, India, etc.

Unfortunately many patients are not diagnosed correctly as alcoholics by their doctors. A high index of suspicion is important, particularly in cases where there are repeated consultations for vague symptoms or minor accidents. If in doubt, a drinking history should be taken in which the patient is asked to describe a typical week's drinking.

Consumption should be quantified in terms of units of alcohol. One unit contains approximately 8 to 10gm of alcohol and is the equivalent of half a pint of beer, a single measure (30ml) of spirits, or a glass of table wine, (**Table 8.5**). Current opinion suggests that drinking becomes a problem at levels above 21 units/week for men and 14 units/week for women.

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Table 8.5 Alcoholic Beverages (Ethanol content per conventional measure)

Beverage	Ethanol Quantity per Measure (in Gm)	Units of Ethanol per Measure
Beer		
Ordinary	16/pint	2/pint
Strong	32/pint	4/pint
Extra-Strong	40/pint	5/pint
Wine		
Table Wine	8/glass	1/glass
Fortified Wine	8/measure	1/measure
	120/bottle	15/bottle
Distilled Spirits	8–12/measure	1–1.5/measure
	240/bottle	30/bottle

Adapted from the **Journal of Royal College of Physicians** (1987, p6).

Medical complications of alcoholism:

1. *GIT* – gastritis, periodic diarrhoea, increased incidence of oropharyngeal and oesophageal cancer.
2. *Liver* – fatty liver with portal hypertension, hepatitis, cirrhosis, increased incidence of hepatic carcinoma.
3. *Pancreas* – acute or chronic pancreatitis.
4. *CVS* – cardiomyopathy, dysrhythmias, hypertension.
5. *CNS* – polyneuropathy, cerebellar degeneration, demyelination of corpus callosum (Marchiafava-Bignami disease), amblyopia, stroke.
6. *RS* – aspiration pneumonia, alcohol-induced asthma.
7. *Endocrine* – hypogonadism and feminisation in males, amenorrhoea, menorrhagia, and infertility in females, pseudo-Cushing syndrome.
8. *Blood* – anaemia, thrombocytopaenia.
9. *Skeletal muscle* – myopathy.
10. *Neuropsychiatric* – memory disturbances (amnesia, blackout), delusions, delirium tremens, Wernicke's encephalopathy, Korsakoff's psychosis, dementia, alcoholic hallucinosis.

Sudden cessation of alcohol intake in a chronic alcoholic can provoke a **withdrawal reaction** which may manifest as one of the following:

1. **Common abstinence syndrome -**
   Onset: 6 to 8 hours after cessation of alcohol.
   Features: Tremor affecting hands, legs, and trunk ("the shakes"), agitation, sweating, nausea, headache, insomnia.

2. **Alcoholic hallucinosis -**
   Onset: 24 to 36 hours.
   Features: Objects appear distorted in shape, shadows seem to move, shouting or snatches of music may be heard.
   Treatment: Administration of phenothiazines (e.g., chlorpromazine 100 mg, 8th hourly).

3. **Seizures (Rum fits) -**
   Onset: 7 to 48 hours.
   Features: Clonic-tonic movements, with or without loss of consciousness.

4. **Alcoholic ketoacidosis -**
   Onset: 24 to 72 hours.
   Features: Occurs during withdrawal as well as after episodes of heavy drinking.[9] In many cases there is a preceding history of g.i disturbance such as gastritis or pancreatitis which has led to sudden diminution of alcohol intake. To compensate for the loss of carbohydrates and depleted glycogen stores, the body mobilises fat from adipose tissue as an alternative source of energy. There is a corresponding decrease in insulin and an increased scretion of glucagon, catecholamines, growth hormone, and cortisol. Fatty acids are oxidised and the final product, acetylcoenzyme A is

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converted to acetoacetate. This in turn is converted to beta-hydroxybutyrate because of ethanol-induced low redox state. Clinical features include drowsiness, confusion, tachycardia, and tachypnoea, progressing to Kussmaul breathing pattern and coma.

Diagnosis: a) Blood alcohol concentration is typically not high. b) Blood glucose may be slightly elevated. c) Since the nitroprusside reaction detects only acetone and acetoacetate and not beta-hydroxybutyrate, the assay for ketones is likely to be only weakly positive. d) There is an elevated anion-gap metabolic acidosis. e) Serum ketones are markedly elevated. f) Hypokalaemia and hypochloraemia are often present.

Treatment: a) Correction of volume depletion – infusion of solutions of normal saline with dextrose. b) Potassium supplementation may be required. c) Thiamine (50 to 100 mg) to prevent development of Wernicke-Korsakoff syndrome.

5. **Delirium tremens (DTs) -**
Onset: 3 to 5 days.
Features: There is a dramatic onset of disordered mental activity characterised by clouding of consciousness, disorientation, and loss of recent memory. There are vivid hallucinations which are usually visual, but sometimes auditory in nature. There is severe agitation with restlessness and shouting, tremor, and truncal ataxia. Insomnia is prolonged. Autonomic disturbances include sweating, fever, tachycardia, hypertension, and dilated pupils. Dehydration and electrolyte disturbances are characteristic. Blood testing shows leucocytosis and impaired liver function.

Treatment: a) Well lit, reassuring environment. b) For agitation diazepam 10 mg i.v initially, and then 5 mg every 5 minutes until full control, followed by 5 to 10 mg orally 3 times daily. c) Thiamine in the usual dose. d) Correction of fluid and electrolyte imbalance.

6. **Wernicke- Korsakoff syndrome -**
This is very rare as a withdrawal phenomenon, and is actually the result of thiamine deficiency due to impoverished diet in an alcoholic.

Features: Wernicke's encephalopathy is the acute form and is characterised by drowsiness, disorientation, amnesia, ataxia, peripheral neuropathy, horizontal nystagmus, and external ocular palsies. When recovery from Wernicke's encephalopathy is incomplete, a chronic amnesic syndrome develops called Korsakoff's psychosis which is characterised by impairment of memory and confabulation (falsification of memory).

Treatment: For Wernicke's encephalopathy – administration of thiamine 50 to 100 mg i.v daily, infused slowly in 500 ml of fluid for 5 to 7 days, and fluid replacement.

Treatment of Ethanol Poisoning

A. **Acute Poisoning:**
1. Airway protection, ventilatory support.
2. Stomach wash.
3. Thiamine 100 mg i.v.
4. 50% dextrose (50 to 100 ml) i.v.
5. Intravenous fluids.
6. A variety of drugs have been tried to hasten the elimination of ethanol or reverse its intoxicating effects, including naloxone, physostigmine, and caffeine. None of them have been proved to be truly effective. Recently, flumazenil (3 mg i.v) has been shown to be effective (in experimental studies) in reversing the respiratory depression associated with ethanol ingestion.

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B. **Chronic Poisoning :**

1. **Treatment of withdrawal** - apart from the treatment measures outlined earlier (*vide supra*), the following drugs have also been tried with varying degrees of success :
   a) Carbamazepine – It has been shown to be effective in treating alcohol withdrawal, including delirium tremens.[11]
   b) Chlormethiazole – It is one of the most popular drugs used for alcohol withdrawal abroad and is administered in a rapidly reducing dosage over 6 to 7 days.
   c) Clonidine has also shown promising results in the treatment of withdrawal symptoms. It is given at a dose of 60 to 180 mg/hr i.v.

2. **Aversion therapy** – The main aim in the treatment of alcoholism is to gradually wean away the patient from the clutches of ethanol, once the acute manifestations of withdrawal have been taken care of. This process referred to quite loosely as de-addiction or detoxification, should be undertaken only after admission to a hospital over a period of several days, under close medical supervision. The insatiable craving for alcohol that is often present must be tackled effectively, and usually requires strongly deterrent measures. Many methods have been tried in this connection, and one of the more succesful ways is to administer a drug called **disulfiram.*** It is a disulfide molecule (tetraethylthiuram) which interferes with the oxidative metabolism of ethanol at the acetaldehyde stage, as a result of which acetaldehyde accumulates producing unpleasant symptoms (**Table 8.6**).

Several investigators now believe that these effects are produced by metabolites of disulfiram (diethyldithiocarbamate and carbon disulfide) and not by acetaldehyde. Whatever the mechanism, the patient avoids intake of alcohol as long as he is on disulfiram.

Principles of disulfiram therapy -

- Ensure that the patient is off alcohol for a minimum period of 12 hours before starting therapy.
- Administer disulfiram only by the oral route.
- Warn the paient explicitly that while he is on disulfiram, alcohol must not be consumed even in small quantity since it can provoke a severe (and sometimes fatal) reaction.
- The usual dose of disulfiram is 250 mg/day which may have to be taken for an indefinite period of time. Such chronic use unfortunately often produces side effects like halitosis (rotten egg odour due to sulfide metabolites), pruritis, headache, drowsiness, impotence, peripheral neuropathies, depression, mania, psychosis, and hepatotoxicity.

Other than disulfiram, there are numerous other substances which evoke a similar reaction with ethanol (**Table 8.7**)

Chronic alcoholism has also been treated with the long acting opiate antagonist naltrexone which is said to reduce the craving for alcohol. Acamprosate is a recent entrant which is claimed to be even more effective in reducing such craving.

*Available in India as **Antadict (Intas), Esperal (Torrent), and Disulfiram (Intas), all tablets of 250 mg strength.**

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Table 8.6 Disulfiram-Ethanol reaction

GIT	CNS	CVS	Skin	RS
Abdominal pain Nausea Vomiting	Blurred vision Confusion Vertigo Headache Weakness	Syncope Hypotension Tachycardia Arrhythmias Chest pain	Sweating Flushing Pruritis	Tachycardia

Table 8.7 Substances producing disulfiram-like reaction with ethanol

Pharmaceuticals		Chemicals	Miscellaneous
Antimicrobials	**Other Drugs**	Calcium cyanamide	Activated charcoal
Cephalosporines	Chlorpropamide	Carbon di sulfide	Mushrooms (Coprinus, Clitocybe)
Chloramphenicol	Glipizide	Hydrogen sulfide	
Furazolidone	MAO Inhibitors	Tetra ethyl lead	
Griseofulvin	Tolbutamide	Tri & Tetra chloroethylene	
Metronidazole			
Nitrofurantoin			

3. **Supportive psychotherapy** – More than individualised psychotherapy, it is group therapy which is effective in the long term management of abstinence. Groups provide an opportunity for resocialisation and a sense of mutual commitment. Self-support organisations such as **Alcoholics Anonymous (AA)** play an important role. The AA which had its origins in the USA in 1935 now has more than 53,000 groups spread worldwide including India. The only requirement for membership is a "desire to stop drinking". There is no membership fee and the organisation functions on a self-supporting basis through contributions from the members. Local addresses of AA groups functioning in a given region can be located in the telephone directory of that area. Meetings are generally held once a week and are informal affairs conducted in a friendly atmosphere. Generally two or three speakers share their experiences during each session relating to their addiction and recovery.

Autopsy Features

1. Congested conjunctivae.
2. Characteristic odour in the vicinity of the mouth and nose, and in the gastric contents.
3. Congestion of g.i tract.
4. Pulmonary and cerebral oedema.
5. Stigmata of chronic alcoholism may be present (fatty or cirrhotic liver, cardiomyopathy, and characteristic lesions in other organs).

Chemical analysis of viscera and body fluids: Apart from the routine viscera and body fluids, one half of brain (or one cerebral hemisphere), as well as samples of CSF and vitreous humor should be collected and preserved for chemical analysis.

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Blood should always be collected from a peripheral vein such as femoral vein and never directly from the heart. Caution must be exercised in interpreting the results of chemical analysis conducted on putrefying dead bodies since postmortem production of ethanol occurs in such cases due to the action of certain microorganisms.[12] This can result in a BAC of upto 20 to 30 mg/100 ml even when no alcohol had been consumed at all prior to death. In such cases the urine should be mandatorily tested, and if no alcohol is detected it can be safely presumed that the BAC is false due to postmortem production.

While blood is the sample of choice when relating the ethanol concentration to the degree of intoxication of the individual before death, it is necessary on occasions to resort to the use of other biological fluids and tissues if it is not possible to obtain a satisfactory sample of blood. As far as urine is concerned, conversion factors have been developed to convert urine alcohol concentration (UAC) to blood alcohol concentration (BAC), but they are not very helful in practice

Forensic Significance

The use or abuse of ethanol is associated frequently with medicolegal implications and consequences. Alcohol abuse is prevalent worldwide. Efforts have been made to totally prohibit alcohol consumption in different countries without much success (with the possible exception of those countries where religious law is in force). Such attempts usually lead to illicit brewing and consumption with even more serious consequences. At present in India, total prohibition is in force only in Gujarat. Efforts to implement the same in Tamilnadu, Andhra Pradesh, and Haryana met with dismal failure.

All over India (except Gujarat where prohibition is in force), the mere consumption of alcohol and consequent drunken behaviour in themselves do not constitute any offence. Only when the drunken individual behaves in such a manner as to become a public nuisance, or poses a threat, or actually commits an act endangering the life or property of another person (or even his own), will the law take cognisance of the fact.

Drunkenness

"My kid's a punk, and I'am a drunk, this I'am sure you will see. Yeah you dont know, what its like to live in misery."

- Neighbour : **Ugly Kid Joe (1991).**

A doctor is sometimes called upon by the police to examine an individual who has been taken into custody for creating public annoyance in a state of intoxication, or for driving a vehicle under the influence of alcohol, or for being involved in a criminal offence after drinking. In such a case, the doctor must exercise great caution before coming to a conclusion. He must first be familiar with the exact meaning of the term '**drunkenness**". An individual can be certified to be drunk only when there is evidence that "*he was so much under the influence of alcohol as to have lost control of his faculties to such an extent as to render him unable to execute safely the occupation in which he was engaged at the material time.*" *The essence of this definition is that the doctor must search for evidence indicating that the individual concerned was under the influence of alcohol. The smell of ethanol in the breath, the pulse rate, dilatation of pupils, colour of the face, etc., give no indication of the degree of intoxication. Conclusion regarding this must be arrived at after a detailed examination with special reference to the state of mind, coordination of movements, visual acuity, etc.

*Based on the report of the **Special Committee of the British Medical Association, 1927**, on drunkenness.

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As in every medicolegal case, before embarking on the physical examination of the individual, the doctor must first obtain his written consent. This must be tempered by the fact that if an individual really is drunk then he is probably in no fit medical condition to give or refuse consent. In such cases, the doctor can proceed with the examination but should not divulge the results or his conclusions until the individual has sobered up and provides valid consent. If an individual refuses consent and is uncooperative, the doctor can proceed with the examination without bothering about this formality if the former has been arrested for a criminal offence and is under the custody of the police (**S. 53 (1), CrPC**). But the requisition in such a case should have been issued by a police officer not below the rank of a Sub-Inspector. The doctor can conduct not only the physical examination of the accused, but can even collect samples of urine or blood for analysis.

Procedure of Examination: **Box 8.2.**

Sometimes the question is asked (when it is proved that a person had consumed alcohol), as to how much he could have drunk, i.e., the quantity of liquor consumed. A rule of thumb is that every ounce of an 80° proof liquor will raise the BAC by 25 mg% (i.e., one peg of distilled spirit, or one glass of wine, or a quarter to half bottle of beer). Average elimination rate is 15 to 20 mg% per hour. As an alternative or supplement to BAC, the level of alcohol can also be estimated in the urine (UAC). To calculate the BAC from the UAC, multiply the latter by 0.66 which gives a rough estimate. For example, if the UAC is 100 mg%, the BAC would be 100 x 0.66 = 66 mg%. It is to be noted however that the first sample of urine voided after the individual was taken into custody by the police must not be used for analysis since there is a likelihood of erroneous interpretation. The person should be asked to empty his bladder and then a second sample is collected after some time which is used for analysis.

Ethanol and Crime

Brawls, assaults (sexual and non-sexual), homicides, and suicides are commonly associated with intoxication. Even if the assailant was so drunk that he had lost total control over his mental faculties and therefore was not in a position to judge whether his actions were right or wong, he is still punishable for any criminal act committed by him (**S. 86, IPC**), unless the alcohol was administered to him without his knowledge or against his will (**S.85, IPC**).

Ethanol and Traffic Accidents

It is well known that consumption of ethanol and consequent intoxication has adverse effects on the driver of a vehicle in the form of visual blurring, motor incoordination, impairment of judgement, and increased reaction time.[13] This has given rise to the adage "Drinking and driving don't mix". Driving a vehicle on a public thoroughfare under the influence of alcohol (or any other intoxicating drug) is an offence in almost every country of the world. In India it is an offence punishable under **Section 185** of the **Motor Vehicle Act** (1988, Amended 1994) in the form of a fine which can extend upto Rs.2000, or imprisonment which can extend upto 6 months, or imposition of both. For a second offence committed within 3 years of the previous one, the punishment is enhanced to Rs.3000, or 2 years, or both. Many countries permit mild degrees of intoxication, but beyond a certain statutory BAC, it becomes a culpable offence. For e.g., the statutory limit is 20 mg% in Poland and Sweden, 50 mg% in Finland, Norway, and Netherlands, 80 mg% in Denmark, Germany, UK, France, and Switzerland, 100 mg% in Ireland, and 80 to 150 mg% in the different states of the USA. In India, the statutory limit has been fixed at 30 mg%. But the general practice is to convict only on the basis of a medical examination, i.e., when it is proved that the driver of the vehicle was under the influence.

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Breathalyser* - In many countries including India, traffic police carry special equipment in the form of breathalysers to detect alcohol in the breath of a suspect driver. It serves as an "on the spot test." Estimates of BAC based on breath analysis are legally admissible as per **S.185** of the **Motor Vehicles Act, 1988** (amended 1994).

In the earliest type of breathalyser, the individual concerned was asked to blow into a plastic balloon containing a crystalline dichromate-sulfuric acid mixture. If the BAC was above a certain prescribed limit, the crystals would turn green to a predetermined distance. However today this has been supersded by more sophisticated versions based on fuel-cell sensing, electrochemical oxidation, infrared photometry, and microprocessors which accurately predict the BAC. From numerous experiments conducted it is now well established that there is an excellent corelation between the breath and blood level, and the ratio is generally 2100 : 1. This is based on **Henry's law** which states that when a volatile chemical (ethanol) is dissolved in a liquid (blood) and is brought to equilibrium with air (alveolar breath), there is a fixed ratio between the concentration of the volatile compound (ethanol) in air (alveolar breath) and its concentration in the liquid (blood), and the ratio is constant at a given temperature, i.e., in this case 34°C which is the temperature at which the breath usually leaves the mouth.[14] But there can be some variation in the ratio between different individuals which is one of the sources of error in the use of breathalysers.

Ethanol and Medical Practice

The medical profession is generally considered to be one of the noble professions and it therefore behoves a doctor to conduct himself at all times with decency and decorum. Although he can use his discretion with reference to moderate consumption of alcohol when not on hospital duty, or when he is not dealing with patients, he can be held liable in the following situations[15] :

1. A surgeon performing an operation under the influence of alcohol can be prosecuted under **S.304-A** of the **IPC** if the patient dies during the course of surgery. The fact that he was drunk at the time of the operation is likely to be considered as strong evidence of rashness.

2. A doctor may be sued for damages in the event of the patient suffering damage or death due to his negligent conduct. If he was intoxicated at the time he administered treatment it would only strengthen the evidence against him.

Patient management under the influence of alcohol will be considered as **infamous conduct (professional misconduct)**, and the doctor is liable to be proceeded against by the State Medical Council. The drunken doctor runs the risk of his name being erased from the medical register.

Box 8.2: Scheme of Examination for Certification of Drunkenness

Objective:

To decide whether

1. The subject is under the influence of alcohol,
2. His condition is due to illness or injury,
3. It is safe for him to be detained in a police station, or he should be admitted to a hospital.

Consent:

Obtain informed, written consent.

Preliminary Particulars :

1. Name, age, sex, occupation, and residential address.

** Also referred to as alcometer, Intoximeter, or drunkometer*

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2. Identification marks : record at least two permanent marks of identification as for any medicolegal case.
3. Time of commencement of examination, (and subsequently), the time of conclusion.
4. Escorts : record the name (and number) of escorting police constable, and his police station.
5. History : a) Has he consumed alcohol ? If so, at what time, and what was the nature and quantity consumed? ; b) Does he suffer from any disease or disability ? ; c) Is he taking any medication or drugs ? If so, what is the nature and dose ?

Physical Examination

1. *General appearance:* state of clothing, behaviour, disposition, etc.
2. *Speech:* normal or slurred or overprecise.
3. *Breath:* odour of alcohol present or absent.
4. *Stance:* does he sway when standing erect with his feet together and eyes closed ?
5. *Gait:* ask him to walk across the room and observe whether the gait is normal or staggering.
6. *Writing:* ask him to write or copy a few sentences in the language with which he is familiar. Note the time taken, repitition or omission of words, etc.
7. *Eyes and visual acuity:* note whether the eyes are reddened, presence or absence of nystagmus, status of pupils, and test for pupillary reaction to light.
8. *Vital data:* record the pulse, temperature, and blood pressure.
9. *Reflexes:* normal or depressed.
10. *Muscular coordination:* test this by asking the individual to perform a few simple tests -
    a) Finger-nose test.
    b) Buttoning/unbuttoning shirt
    c) Picking up pencil/pen from the floor.

Examine also the cardiovascular, pulmonary, and alimentary systems in the usual way. Look for the presence of injuries.

Mental Examination

1. *Memory:* test for ability to recall recent events.
2. *Orientation: to time* - ask him to state the approximate time of day, or the day of the week; *to space* - ask him as to whether he is aware of his present location.

Laboratory Investigations

Collect blood and urine samples. The collected sample of blood must be preserved in a chemically clean container with sodium fluoride as preservative (50 mg for 5 ml). Some workers believe that it is desirable to add potassium oxalate (15 mg). For urine sample, the recommended preservative is phenyl mercuric nitrate.

Opinion

This should be phrased in one of the following ways:

a) The individual examined has not consumed alcohol.
b) The individual examined has consumed alcohol, but is not under its influence.
c) The individual examined has consumed alcohol and is under its influence.

Opinion (a) is given if there is no smell of alcohol in the breath and/or laboratory analysis is negative for alcohol; and all the clinical findings are normal; **Opinion (b)** is given if there is smell of alcohol in the breath and/or laboratory analysis reveals the presence of alcohol; but clinical examination reveals only normal findings; **Opinion (c)** is given if there is smell of alcohol in the breath and/or laboratory analysis reveals the presence of alcohol; and clinical examination reveals abnormal findings.

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Authentication
Signature of the medical officer, his name, and designation.

Methanol

Synonyms
Methyl alcohol, wood spirit, wood naphtha, colonial spirit

Physical Appearance
Clear, colourless, volatile liquid with characteristic odour and bitter taste.

Uses & Sources
1. Antifreeze (10 to 50%)
2. Denatured spirit (5 to 10%)
3. Embalming fluid (20%)
4. Leather dyes (30%)
5. Varnish & shellac (5%)
6. Windshield washing fluid (35 to 95%)

Usual fatal Dose
70 to 100 ml (Range 15 to 250 ml).

Mode of Action
In the liver, methanol is metabolised to formaldehyde (by alcohol dehydrogenase) and then to formic acid (by aldehyde dehydrogenase) which is responsible for retinal toxicity as well as metabolic acidosis.

Clinical Features
Symptoms may be delayed for 12 to 24 hours. The earliest manifestations include vertigo, headache with stiff neck (meningismus), nausea, vomiting, and abdominal pain.

Later there is ocular toxicity characterised by blurred or dimmed vision ("flashes" or "snowstorm"), and photophobia. Ophthalmologic examination usually reveals dilated pupils with sluggish light reaction. Fundoscopy reveals hyperaemia of optic disc followed by retinal oedema. Irreversible sequelae include optic atrophy and visual field impairment.

Metabolic acidosis (high anion gap) is usually severe. Other features include tachycardia, hypotension, and hypothermia. Convulsions are a late feature and may be followed by coma.

Cause of death is usually respiratory failure, which may precede the cessation of heart beat by several minutes.

Diagnosis
1. High anion gap acidosis.
2. Elevated osmolal gap.
3. Blood methanol level : more than 50 mg / 100 ml indicates serious poisoning.

Treatment
1. Stomach wash with sodium bicarbonate.

2. **Ethanol** is the specific antidote since it preferentially competes for the same enzyme (alcohol dehydrogenase) and prevents the metabolism of methanol which is then excreted unchanged in the urine.

   Mode of administration: 10% ethanol * at a dose of 10 ml/kg administered i.v over 30 minutes, followed by 1.5 ml/kg/hr, so as to produce and maintain a blood ethanol level of 100 mg/100 ml. Alternatively, 1 ml/kg of 95% ethanol in fruit juice (180 ml) can be given orally over 30 minutes. For maintenance, administer 0.17 to 0.28 ml/kg/hr as 50% ethanol in fruit juice.

To prepare 10% ethanol for i.v use, remove 100 ml from 1 litre of 5 % dextrose and substitute with 100 ml of absolute alcohol after passing through filter paper (0.22u).

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In Western countries, a new antidote has been introduced viz., **4 methyl pyrazole** (4MP), or **fomepizole** which does not cause CNS depression (unlike ethanol).

3. Sodium bicarbonate i.v : 500 to 800 ml of 7.5% solution, slowly.

4. Folinic acid i.v : 1 to 2 mg/kg, 6th hourly. It hastens the elimination of formic acid.

5. Haemodialysis is very effective in removing methanol, formaldehyde, and formic acid.[fo] Haemoperfusion is not effective.

Autopsy Features

Cyanosis is very prominent, especially in the upper parts of the body. Liver and kidneys show toxic damage. Lungs may reveal oedema, emphysematous changes, and desquammation of alveolar epithelium. Eyes show evidence of retinal oedema.

Viscera must be preserved in saturated solution of sodium chloride and not rectified spirit, as in the case of all alcohols. In addition to the routine viscera, it is advisable to preserve one cerebral hemisphere.

Forensic Significance

Most of the cases of methanol poisoning are accidental arising out of either an alcoholic (deprived of ethanol for any reason) consuming methanol containing products, or because of intentional adulteration of ethanol (especially arrack) resulting in mass deaths. The latter are referred to quaintly as "liquor tragedies" and are reported in Indian newspapers at depressingly regular intervals from all parts of the country.

Isopropanol

Synonyms

Isopropyl alcohol, 2-propanol, Blue heaven*

Physical Appearance

Colourless, volatile liquid with a faint odour of acetone, and a slightly bitter taste.

Uses

1. Rubbing alcohol (70%), for massage.
2. Disinfectant.
3. Paint remover.
4. Toiletries (hair tonics, after-shave lotions).
5. Industrial solvent.

Usual Fatal Dose

250 to 300 ml. In terms of blood level:> 300 mg%.

Toxicokinetics

Isopropanol can be absorbed through all routes. In the body it is rapidly metabolised by alcohol dehydrogenase. Approximately 80% is converted to acetone, and the remainder is excreted unchanged in the urine. Acetone is excreted in the urine and breath.

Mode of Action

Isopropanol is two to three times more potent than ethanol as a CNS depressant.

Clinical Features

Lethargy, vertigo, headache, confusion, ataxia, miosis, abdominal pain, gastritis, haemorrhagic tracheobronchitis, hypotension, and apnoea. It can also cause haemolytic anaemia, myopathy, and acute renal failure.

A characteristic odour of acetone is usually perceptible in the breath.

**In hospitals, isopropanol is often coloured blue to distinguish it from other colourless liquids, which has led to the designation "blue heaven" by abusers.*

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Diagnosis
1. Ketonuria.
2. Increased osmolal gap.
3. High anion gap metabolic acidosis.

Treatment
1. Skin decontamination in the case of dermal exposure.
2. Stomach wash and activated charcoal in the case of ingestion.
3. Haemodialysis.
4. Supportive measures, including correction of hypotension, metabolic acidosis, etc.

Ethylene Glycol

Physical Appearance
Colourless, syrupy, odourless, non-volatile liquid, with a bitter-sweet taste.

Uses
1. Antifreeze
2. Coolant
3. Hydraulic brake fluid

Usual Fatal Dose
70 to 100 ml

Mode of Action
Ethylene glycol is not absorbed through skin, and because of its low vapour pressure does not produce toxicity upon inhalation. It is however absorbed rapidly through the g.i tract and is metabolised (more than 80%) to glycoaldehyde, glycolic acid, and oxalic acid which inhibit diverse metabolic pathways in the body, including oxidative phosphorylation.

Clinical Features
First Phase (CNS stage) : upto 12 hours post-ingestion.

This stage is mainly due to the parent compound itself and is characterised by vomiting, inebriation, lethargy, nystagmus, ataxia, convulsions, and coma.

Second Phase (CVS stage): 12 to 24 hours post-ingestion.

This stage is characterised by tachycardia, tachypnoea, congestive heart failure, and circulatory collapse.

Third Phase (Renal stage): 24 to 72 hours post-ingestion.

There is oliguria, flank pain, acute tubular necrosis and renal failure. Urine contains calcium oxalate or hippurate crystals. Hypocalcaemia results in manifestations of tetany.

Diagnosis
1. High anion gap acidosis.
2. Osmolal gap.
3. Calcium oxalate crystals in urine.

Treatment
Do not wait for symptoms to appear
1. Stomach wash and activated charcoal.
2. **Ethanol** is the antidote and must be given i.v., (same as for methanol poisoning). It inhibits the metabolism of ethylene glycol. **4-methyl pyrazole** is a better alternative.
3. Haemodialysis.
4. Sodium bicarbonate i.v.
5. Pyridoxine 50 mg and thiamine 100 mg i.m., 6th hourly for 2 days.
6. Monitor serum calcium level and replace as indicated, with 10% calcium gluconate i.v.

Autopsy Features
1. Cerebral oedema, chemical meningoencephalitis.
2. Toxic damage of liver and kidneys.
3. Oxalate crystals in brain, spinal cord, and kidneys.

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BENZODIAZEPINES

Examples

Alprazolam, chlordiazepoxide, clonazepam, diazepam, flurazepam, lorazepam, midazolam, nitrazepam, oxazepam, and triazolam.

Uses

Management of

- Anxiety disorders
- Seizure disorders
- Insomnia
- Movement disorders (adjunctive therapy)
- Mania (adjunctive therapy)

Toxicokinetics

Most benzodiazepines are administered orally or by i.v injection. Following absorption, all benzodiazepines are bound to plasma proteins to the extent of 70 to 99%, and are metabolised extensively by different microsomal enzyme systems in the liver.

Mode of Action

Benzodiazepines act by stimulating the $GABA_b$ (g-aminobutyric acid b) receptors, thereby opening up the chloride ion channel in the receptor complex, resulting in the increased conductance of chloride ion across the nerve cell membrane. This lowers the potential difference between the interior and exterior of the cell, blocking the ability of the cell to conduct nerve impulses.[17].

Adverse Effects

Weakness, headache, amnesia, vertigo, diplopia, nausea, diarrhoea, and rarely chest pain. Paradoxical effects (disinhibition or dyscontrol reaction) may sometimes occur characterised by restlessness, agitation, and hallucinations. Triazolam is noted for its tendency to produce delirium and toxic psychosis and has been banned in the United Kingdom since 1991.

Toxic Effects

Benzodiazepines are remarkably safe drugs and rarely produce serious toxic effects even with substantial ingestion. Death is uncommon unless other synergistic drugs have also been ingested.[18]

Acute Poisoning:

Mild – Drowsiness, ataxia, weakness.

Moderate to Severe – Vertigo, slurred speech, nystagmus, partial ptosis, lethargy, coma. Hypotension and respiratory depression supervene in potentially lethal ingestions.

Chronic Poisoning:

Long term use of benzodiazepines is associated with the development of tolerance. Abrupt cessation provokes a mild withdrawal reaction characterised by anxiety, insomnia, headache, tremor, and paraesthesia. Restlessness, encephalopathy, and hallucinations may occur after abrupt withdrawal from high daily doses.[19].

Diagnosis

Gas chromatography-mass spectrometry (GC-MS) can be used to analyse urine levels of benzodiazepines. A less effective alternative is thin layer chromatography (TLC), which can be done on urine, gastric aspirate, or scene residue. Estimation of plasma levels of benzodiazepines is usually not necessary.

Treatment

Acute Poisoning:

- Decontamination – Stomach wash may be helpful if the patient is seen within 6 to 12 hours. Activated charcoal adsorbs benzodiazepines and can be administered in the usual manner.
- Establish clear airway. Oxygen and assisted ventilation are often necessary.
- I.V fluids.
- Correction of hypotension with dopamine or levarterenol.

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- Antidote – **Flumazenil** is effective in reversing the coma induced by benzodiazepines. The effect is however usually short-lived, and flumazenil also has the tendency to induce a withdrawal reaction in benzodiazepine-dependent patients. The mode of action is competitive antagonism. In practice, most patients achieve complete reversal of benzodiazepine effect with a total slow i.v dose of just 1 mg.[20] Some investigators suggest that flumazenil is better administered in a series of smaller doses in an incremental manner beginning with 0.2 mg and progressively increasing by 0.1 to 0.2 mg every minute until a cumulative total dose of 3.5 mg is reached.[21]

  However, re-sedation occurs within ½ hour to 2 hours (depending on the nature and dose of benzodiazepine ingested), and therefore patients must be carefully monitored and subsequent doses of flumazenil should be administered as needed.

Chronic Poisoning:

- Phenobarbitone-substitution technique is recommended for benzodiazepine withdrawal which employs propranolol for acute somatic symptoms, while phenobarbitone is used for detoxification.[22] However the most frequently used method among clinicians is the replacement of a short half-life benzodiazepine (such as alprazolam) with a long half-life benzodiazepine (such as clonazepam), before initiating a taper and final discontinuation.

Forensic Significance

Ever since the introduction of benzodiazepines in the 1960s, they have become progressively more popular as anxiolytic agents and sedatives, displacing the barbiturates from their previously held top spot. In spite of extensive use worldwide, there have been only a few cases reported involving fatalities, demonstrating the wide margin of safety of benzodiazepines. But complacency must be avoided and the safety profile of these drugs should not be taken for granted, as deaths have been reported in some recent cases even from unexpectedly low doses of certain benzodiazepines.[23] There are also indications that some of the newer benzodiazepines have a slightly smaller margin of safety. Yet another important issue is with reference to the use of benzodiazepines to deliberately induce amnesia in certain individuals in order to accomplish an immoral act (e.g., *date rape*). Many of these drugs are capable of causing anterograde amnesia.

While addiction to benzodiazepines is an undeniable possibility among patients on long term therapy, the abuse potential is much less when compared to most other sedative-hypnotics such as the barbiturates.

BARBITURATES

The barbiturates are derivatives of barbituric acid (2,4,6-trioxohexahydropyrimidine) and were extensively used as sedative-hypnotics till the 1960s when the benzodiazepines arrived and quickly displaced them.

Examples

Listed in **Table 8.8**.

Uses

- Sedative-hypnotic.
- Pre-operative sedation.
- Treatment of seizure disorders.

Toxicokinetics

Most barbiturates which are used as sedative-hypnotics are administered orally. Intravenous route is usually reserved for management of status epilepticus or induction/maintenance of general anaesthesia.

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Following absorption, barbiturates are distributed widely. Metabolism of most of these drugs occurs by oxidation in the liver resulting in the formation of alcohols, ketones, phenols, or carboxylic acids which are excreted in the urine as such or in the form of glucuronic acid conjugates. About 25% of phenobarbitone is excreted unchanged in the urine.

Table 8.8 Classification of Barbiturates

Ultra-short acting (duration of action < 15-20 min) Thiopentone Methohexitone
Short acting (duration of action < 3 hrs) Hexobarbitone Pentobarbitone Secobarbitone
Intermediate acting (duration of action 3-6 hrs) Amobarbitone Aprobarbitone Butobarbitone
Long acting (duration of action 6-12 hrs) Mephobarbitone Phenobarbitone

Adverse Effects

- Residual depression after the main effect of the drug has passed off.
- Paradoxical excitement (especially in the elderly).
- Hypersensitivity reaction – localised swelling of eyelid, cheek, or lip, erythematous or exfoliative dermatitis.
- Synergistic action with ethanol and antihistamines.

Toxic Effects

1. Slurred speech, ataxia, lethargy, confusion, headache, nystagmus.
2. CNS depression, coma, shock.
3. Pupils are at first constricted, but later dilate because of hypoxia.
4. Hypothermia.
5. Cutaneous bullae (blisters).

Death may occur from respiratory arrest or cardiovascular collapse.

Chronic barbiturate (ab)use is associated with the development of tolerance which is responsible for decreasing the therapeutic to toxic index. An addict may obtain therapeutic benefit only with 5 to 6 times the normal dose ! Abrupt withdrawal results in anorexia, tremor, insomnia, cramps, seizures, delirium, and orthostatic hypotension.

Usual Fatal Dose

Phenobarbitone: 6 to 10 gm.

Amobarbitone, pentobarbitone, secobarbitone : 2 to 3 gm.

Lethal blood level for short- and intermediate-acting barbiturates varies from 3 to 4 mg/100 ml, while for phenobarbitone it ranges from 8 to 15 mg/100 ml.

Diagnosis

- Thin layer chromatography (for urine, stomach contents, or scene residue).
- Gas chromatography or high pressure liquid chromatography.
- EEG: alpha coma indicates poor prognosis.[24]

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Treatment

1. Gastric lavage (preferably with a large-bore, double-lumen tube), can be done with benefit upto 12 to 24 hours post-ingestion.
2. Activated charcoal in the usual dose.
3. Forced alkaline diuresis is said to be particularly useful in phenobarbitone poisoning.
4. Haemodialysis or haemoperfusion.
5. Supportive measures : supplemental oxygen, intubation, assisted ventilation, i.v fluids.

Autopsy Features

1. Peripheral cyanosis.
2. Froth at the mouth and nose.
3. Barbiturate blisters on the dependent parts of skin surface : buttocks, inner aspects or back of thighs, calves, and forearms.
4. Intensely congested lungs.
5. Congestion or even erosion of the stomach.

Forensic Significance

The incidence of poisoning with barbiturates has declined dramatically in recent years as a direct result of decline in their use as sedative-hypnotics. However cases do get reported even today, mostly the result of deliberate self-ingestion. This type of (suicidal) poisoning was rampant in the 1960s when these drugs were widely prescribed and consequently abused. One of the most famous cases during this period concerned immortal Hollywood actress Marilyn Monroe who at the end of her short, tempestuous career became hopelessly addicted to alcohol and barbiturates. In 1962, at the age of 36, Marilyn Monroe was found dead at home following an overdose of barbiturates.

In India, as per some studies, barbiturates used to account for the maximum number of poison-related fatalities every year right upto the early 1970s, but after that the incidence has plummeted rapidly, and today they account for only a negligible proportion of deaths.[25]

Accidental barbiturate poisoning due to inadvertent overdose is not uncommon among addicts because of their phenomenon of tolerance. But earlier hypotheses that a patient could overdose himself to death by so-called *automatic behaviour* brought on by sleepy confusion have not withstood scientific analysis.

Intravenous thiopentone has been used as *truth serum* to extract confessions during interrogation by inducing a state of drowsy disorientation in the course of which the person may reveal the truth.

CHLORAL HYDRATE

Chloral hydrate is rarely used as a hypnotic today, but is a common adulterant of illicit liquor to enhance its intoxicating effect (**Mickey Finn** or **Knock-out drops**). It is a white crystalline substance soluble in water or alcohol with a pungent, pear-like odour and bitter taste. Chloral hydrate is well absorbed on oral administration and is quickly metabolised to trichloroethanol in the liver by alcohol dehydrogenase. This is the active form which is later conjugated with glucuronic acid and excreted in the urine as urochloralic acid.

Chloral hydrate overdose manifests as nausea, vomiting, gastric irritation, miosis, hypotension, renal and hepatic damage, and cardiac arrhythmias. The usual fatal dose is around 10 gm.

Treatment should be mainly directed at the management of cardiac arrhythmias which are potentially life threatening. Unfortunately the arrhythmias are usually non-responsive to conventional anti-arrhythmic drugs, and a beta-adrenergic antagonist (non-cardioselective or $beta_1$-specific), or adrenergic neurone blocking drug such as bretylium may have to be administered. Flumazenil may produce dramatic improvement in chloral hydrate poisoning according to some investigators.[26]

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CHAPTER - 9

Pesticides

Pesticides are compounds that are used to kill pests which may be insects, rodents, fungi, nematodes, mites, ticks, molluscs, or unwanted weeds and herbs.

Classification

I. Insecticides: Compounds which kill or repel insects and related species. Egs : organophosphates, carbamates, organochlorines, diethyltoluamide (DEET), and pyrethrum and its derivatives (pyrethroids).

II. Rodenticides: Compounds which kill rats, mice, moles, and other rodents. Egs : anticoagulants, thallium, vacor, phosphorus, alpha-naphthylthiourea, cholecalciferol, arsenic, barium carbonate, bromethalin, fluoroace-tamide, sodium monofluoroacetamide, red squill, strychnine, and zinc phosphide.

III. Herbicides: Compounds which kill weeds. Egs: acrolein, dalaphon, paraquat, diquat, atrazine, propazine, simazine, nitrofen, trichloroacetic acid, and chlorophenoxy compounds.

IV. Fungicides: Compounds which kill fungi and moulds. Egs: thiocarbamates, captan, captafol, bavistin, vitavax, hexachlorobenzene, and sodium azide.

V. Nematicides: Compounds which kill nematodes (i.e., worms). Egs : ethylene dibromide.

VI. Acaricides: Compounds which kill mites, ticks, and spiders. Egs : azobenzene, chlorobenzilate, tedion, and kelthane.

VII. Molluscicides: Compounds which kill molluscs such as snails and slugs. Egs : metaldehyde.

VIII. Miscellaneous Pesticides: Compounds of lead, copper, and mercury, nicotine, rotenone, sabadilla, hydrogen cyanide, methyl bromide, naphthalene, tetrachloroethylene, trichloroethane, dinitrophenol, dinitrocresol, dinitrobutylphenol, pentachlorophenol, chlorfenson, and chloralose.

Only the following pesticides will be discussed– **organophosphates, carbamates, organochlorines, pyrethroids,** and **strychnine.** An important pest-repellent **aluminium phosphide** has been discussed in an earlier section (page no. 315)

ORGANOPHOSPHATES

They are among the most popular and most widely used insecticides in India. **Table 9.1** lists common varieties along with respective brand names.

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Table 9.1 Common Organophosphate Compounds

Generic Name	Brand Name
Acephate	Acemil, Agrophate, Asataf, Hythane, Starthene
Chlorfenvinphos	Birlane, Chlorfenvinphos
Chlorpyriphos	Agrofas 20, Chlorofos 20, Coroban 20, Dursban, Gilphos, Hyban 20, Ruban 20, Tafaban
Diazinon	Agroziron, Basudin, Bazanon, Ditaf, Suzinon, Zionosul 50
Dichlorvos	Agrovan 76, Agro 76 EC, Bangvas, DDVP, Divap, Divisol, Nuvan, Nuvasul 76, Paradeep, Savious,Vapona, Vapox, Vegfru
Dimethoate	Agrodimet 30, Agromet 30EC, Bangor 30EC, Corothate, Cropgor 30, Cygon, Devigor, Dimethoate, Dimex, Entogor, Hexagor, Hygro 30, Klex Dimethoate, Milgor, Paragor, Parrydimate, Ramgor, Rogor, Tagor, Tara Dimex Sulgor, Vijaygor, Vikagor
Ediphenphos	Hinosan
Ethion	Demite, Dhan-unit, Ethion, Ethiosul 50, Force, Mit 505, Miticil, RP-thion, Tafethion, VegFru Fosmite
Fenitrothion	Accothion, Agrothion, Danathion, Fenicol, Fenitrosul 50, Folithion, Sumithion, Tik 20, Vikathion
Fenthion	Agrocidin, Baytex, Fenthiosul, Lebaycid
Formothion	Anthio
Glyphosate	Weed Off
Malathion	Agromal, Cythion, Finit, Kathion, Licel, Maladan, Malazene, Sulmathion, VegFru Malatox
Methyl Parathion	Agropara, Agrotex, Ekatox, Folidol-M, Harvest Kempar, Kilex-M, Metacid, Metapar, Paracrop, Parahit, Parataf, VegFru Paratox
Monocrotophos	Azodrin, Corophos, Entophos, Macrophos, Microphos, Monocil, Monocron, Monokem, Nuvacron
Oxydemeton Methyl	Hexasystox, Hymox, Knock Out, Metasystox
Phenthoate	Agrofen, Delsan, Elsan, Guard, Phentox
Phorate	Dragnet, Fortan, Glorat, Luphate, Phoratox, Thimet, Volphor, VegFru Foratox
Phosphamidon	Agromidon 85, Bangdon 85, Cildon, Delphamidon, Dimecron, Directon, Entecron 85, Phamidon, Phosul, Sudon, Vimidon
Primiphos Methyl	Acetellic
Quinalphos	Agroquin, Agroquinol, Bayrusil, Ekalux, Kilex, Quinal, Solux, Vikalux
Temephos	Abate 50EC, Farmico's
Thiometon (Morphothion)	Agrothimeton, Ekatin
Triazophos	Hostathion
Trichlorphon	Dipterex

Adapted from Pillay VV. Pocketbook of Pesticide Poisoning for Physicians 2001. CBS Publishers, New Delhi.

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Physical Appearance

These compounds are available as dusts, granules, or liquids. Some products need to be diluted with water before use, and some are burnt to make smoke that kills insects.

Usual Fatal Dose

Toxicity Rating (As per the **Insecticide Rules, 1971**)

The following compounds are **extremely toxic** (LD50 : 1 to 50 mg/Kg), or **highly toxic** (LD50 : 51 to 500 mg/Kg):

Chlorfenvinphos, Chlorpyriphos, Diazinon, Dichlorvos, Dimethoate, Ediphenphos, Ethion, Fenitrothion, Fenthion, Formothion, Methyl Parathion, Monocrotophos, Oxydemeton Methyl, Phenthoate, Phorate, Phosphamidon, Quinalphos, and Thiometon.

The following compounds are **moderately toxic** (LD50: 501 to 5000 mg/Kg), or **slightly toxic** (LD50: more than 5000 mg/Kg): Acephate, Glyphosate, Malathion, Phenthoate, Primiphos Methyl, Temephos, Triazophos, and Trichlorphon.

Toxicokinetics

Organophosphates can be absorbed by any route including transdermal, transconjunctival, inhalational, across the g.i and g.u mucosa, and through direct injection.

Manifestations usually begin within a few minutes to few hours.

Mode of Action

Organophosphates are powerful inhibitors of acetylcholinesterase which is responsible for hydrolysing acetylcholine to choline and acetic acid after its release and completion of function (i.e., propagation of action potential). As a result, there is accumulation of acetylcholine with continued stimulation of local receptors and eventual paralysis of nerve or muscle. Although organophosphates differ structurally from acetylcholine, they can bind to the acetylcholinesterase molecule at the active site and phosphorylate the serine moiety. Phosphorylated enzymes degrade very slowly over days to weeks, making the acetylcholinesterase essentially inactive.

Once the acetylcholinesterase is phosphorylated, over the next 24 to 48 hours an alkyl group is eventually lost from the conjugate, further exacerbating the situation. As this occurs, the enzyme can no longer spontaneously hydrolyse and becomes permanently inactivated.[1].

Clinical Features

Acute Poisoning:

A. *Cholinergic Excess**-

I. Muscarinic Effects (hollow organ parasympathetic manifestations): Common manifestations include bronchoconstriction with wheezing and dyspnoea, cough, pulmonary oedema, vomiting, diarrhoea, abdominal cramps, increased salivation, lacrimation, and sweating, bradycardia, hypotension, miosis, and urinary incontinence.

II. Nicotinic Effects (autonomic ganglionic and somatic motor effects): Fasciculations, weakness, hypertension, tachycardia, and paralysis.

B. *CNS Effects*: Restlessness, headache, tremor, drowsiness, delirium, slurred speech, ataxia, and convulsions. Death usually results from respiratory failure.

** Often summarised in the mnemonic DUMBELS – Diarrhoea, Urination, Miosis, Bronchospasm, Emesis, Lachrmation, Salivation.*

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A characteristic kerosene-like odour is often perceptible in the vicinity of the patient since the solvent used in many organophosphate insecticides is some petroleum derivative such as aromax.

It is important to note that –

- In a given case, there may be either tachy- or bradycardia ; hypo- or hypertension.
- Miosis while being a characteristic feature, may not be apparent in the early stages. In fact mydriasis is very often present, and hence treatment should not be delayed if there is absence of pupillary constriction.[2]
- Ocular exposure can result in systemic toxicity. It can cause persistent miosis in spite of appropriate systemic therapy, and may necessitate topical atropine (or scopolamine) instillation.[3]
- While respiratory failure is the commonest cause of death, other causes may contribute including hypoxia due to seizures, hyperthermia, renal failure, and hepatic failure.
- An **Intermediate Syndrome** sometimes occurs one to four days after poisoning due to long-lasting cholinesterase inhibition and muscle necrosis.[4] Main features include muscle weakness and paralysis characterised by motor cranial nerve palsies, weakness of neck flexor and proximal limb muscles, and acute respiratory paresis. It may be due to inadequate treatment of the acute episode especially involving subtherapeutic administration of oximes or inadequate assisted ventilation. Once it sets in, the intermediate syndrome will have to be managed by supportive measures, since it does not respond to oximes or atropine.
- A **Delayed Syndrome** sometimes occurs 1 to 4 weeks after poisoning due to nerve demyelination, and is characterised by flaccid weakness and atrophy of distal limb muscles, or spasticity and ataxia.[5] This syndrome also does not respond to either oximes or atropine.

Chronic Poisoning:

It usually occurs as an occupational hazard in agriculturists, especially those who are engaged in pesticide spraying of crops. Route of exposure is usually inhalation or contamination of skin. The following are the main features -

1. *Polyneuropathy:* paresthaesias, muscle cramps, weakness, gait disorders.
2. *CNS Effects:* drowsiness, confusion, irritability, anxiety, psychiatric manifestations.

Diagnosis

1. **Depression of cholinesterase activity:**
   a) If the RBC cholinesterase level is less than 50% of normal, it indicates organophosphate toxicity.

      Disadvantages -
      - A very low cholinesterase level does not always corelate with clinical illness.
      - False depression of RBC cholinesterase level is seen in pernicious anaemia, haemoglobinopathies, antimalarial treatment, and blood collected in oxalate tubes.

   b) Depression of plasma cholinesterase level (to less than 50%) is a less reliable indicator of organophosphate toxicity, but is easier to assay and more commonly done. Because it is a liver protein, plasma cholinesterase activity is depressed in cirrhosis, neoplasia, malnutrition, and infections.

      For the purpose of estimation of cholinesterase level, blood should be collected only in heparinised tubes. Alternatively, samples can be frozen.

2. **P-Nitrophenol Test:** P-nitrophenól is a metabolite of some organophosphates (e.g., parathion, ethion), and is excreted in the urine.

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Procedure: Steam distill 10 ml of urine and collect the distillate. Add sodium hydroxide (2 pellets) and heat on a water bath for 10 minutes. Production of yellow colour indicates the presence of p-nitrophenol. The test can also be done on vomitus or stomach contents.

3. **Thin Layer Chromatography** (TLC)

Treatment

1. **Decontamination:**
   a) If skin spillage has occurred, it is imperative that the patient be stripped and washed thoroughly with soap and water.
   b) If ocular exposure has occurred, copious eye irrigation should be done with normal saline or Ringer's solution. If these are not immediately available, tap water can be used.
   d) In the case of ingestion, stomach wash can be done. Activated charcoal is beneficial.

2. **Antidotes:**
   a) *Atropine:* Atropine, a competitive antagonist of acetylcholine at the muscarinic postsynaptic membrane and in the CNS, will block the muscarinic manifestations of organophosphate poisoning.

   b) *Pralidoxime:* This is a nucleophilic oxime which helps to regenerate acetylcholinesterase at muscarinic, nicotinic, and CNS sites.

   The antidotes for organophosphates have been discussed together in detail in **Box 9.1**

3. **Supportive measures:**
   a) Administer i.v fluids to replace losses.
   b) Oxygenation/intubation/positive pressure ventilation.
   c) The following drugs are contraindicated : parasympathomimetics, phenothiazines, antihistamines, and opiates.

4. **Prevention of further exposure**: After the patient has recovered, he should not be re-exposed to organophosphates for at least a few weeks since he is likely to suffer serious harm from a dose that normally would be harmless, owing to alteration of body chemistry.[6]

Autopsy Features[7]

External:

1. Characteristic odour (garlicky or kerosene-like).
2. Frothing at mouth and nose.
3. Cyanosis of extremities.
4. Constricted pupils.

Internal:

1. Congestion of g.i tract ; garlicky or kerosene-like odour of contents.
2. Pulmonary and cerebral oedema.
3. Generalised visceral congestion.

A useful sign in mortuaries infested with flies is that some of these flies after settling on an opened body (dead of organophosphate poisoning), may themselves subsequently die and land on the autopsy table ! It is also pertinent to remember that organophosphates resist decomposition and can be easily detected even in putrefied bodies.

Forensic Significance

Discussed at the end of the section, together with all the other pesticides.

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Box 9.1

Antidotes for Organophosphates

Atropine

Mode of action : Blocks the muscarinic manifestations of organophosphates. However, since atropine affects only the postsynaptic muscarinic receptors, it has no effect on muscle weakness or paralysis.

Dose: 1 to 2 mg i.v or i.m (adult) ; 0.05 mg / kg i.v (child) ; every 15 minutes until the endpoint is reached i.e., drying up of tracheobronchial secretions. Pupillary dilatation and tachycardia are not reliable indicators of the end point. Once the end point has been reached, the dose should be adjusted to maintain the effect for at least 24 hours.[1]

Availability : Atropa inj (Jawa Pharma) of 0.6 mg/ml, 2 ml ampoules ; Tropine inj (Neon Labs) of 0.6 mg/ml, 1 ml ampoules.

Pralidoxime (Pyridine-2-aldoxime methiodide ; 2-PAM)

Mode of action : It is usually given along with atropine. Pralidoxime competes for the phosphate moiety of the organophosphorus compound and releases it from the acetylcholinesterase enzyme, thereby liberating the latter and reactivating it. While it is advisable to begin pralidoxime therapy within 48 hours of poisoning, it can be administered even much later with beneficial effects.[2]

Dose : For adults – 1 to 2 gm in 100 to 150 ml of 0.9% sodium chloride, given i.v over 30 minutes. This can be repeated after 1 hour, and subsequently every 6 to 12 hours, for 24 to 48 hours. Maximum dose should not exceed 12 gm in a 24 hour period.

For children – 20 to 40 mg/kg to a maximum of 1 gm/dose given i.v, and repeated every 6 to 12 hours for 24 to 48 hours.

It is generally not advised for the treatment of carbamate overdose, especially carbaryl.

Availability : PAM tabs, 500 mg, for oral use (Shree Ganesh) ; PAM-A Korea inj of 500 mg in 20 ml, for i.v use (Shree Ganesh) ; PAM-A inj of 500 mg in 20 ml, for i.v use (Panacea).

Diazepam

Some studies indicate that the addition of diazepam to atropine and 2-PAM improves survival.[3] it reduces the risk of seizure-induced brain and cardiac damage.

Dose : For adults – 5 to 10 mg i.v slowly, every 15 minutes, upto a maximum of 30 mg.

For children – 0.25 to 0.4 mg/kg i.v slowly, every 5 to 10 minutes, upto a maximum of 10 mg.

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CARBAMATES

They are as popular as organophosphates in their role as insecticides and share a number of similarities. Only the differentiating features will be discussed. Indian brands are listed in **Table 9.2**.

Usual Fatal Dose

Toxicity Rating (As per the **Insecticide Rules, 1971**)

The following are **extremely toxic** (LD50 : 1 to 50 mg/Kg), or **highly toxic** (LD50 : 51 to 500mg/Kg): Carbaryl, Carbofuran, Methomyl, Propoxur.

The following are **moderately toxic** (LD50 : 501 to 5000 mg/Kg), or **slightly toxic** (LD50: more than 5000 mg/Kg): Aldicarb, Carbendazim, Triallate.

Mode of Action

Carbamates (like organophosphates) are inhibitors of acetylcholinesterase, but carbamylate the serine moiety at the active site instead of phosphorylation. This is a reversible type of binding and hence symptoms are less severe and of shorter duration. With respect to the nature of clinical manifestations, there is general similarity between carbamates and organophosphates.[8].

Table 9.2 Common Carbamates

Generic Name	Brand Name
Aldicarb	Temik
Carbaryl	Agrovin, Agroyl, Bangvin 50, Caravet, Hexavin, Kevin 50, Kilex Carbaryl, Sevin 50, Sujacarb, Sulfarl 50
Carbaryl + Gamma BHC	Sevidol
Carbendazim	Bavistin, Carbistin 50, Gilzim, Kilex Carbendazim 50, Spot Free
Carbofuran	Agrofuron 3G, Carbocil 3, Furadan 3G, Hexafuran, VegFru Diafuran
Methomyl	Lannate
Propoxur	Baygon, Protox Bait
Triallate	Avadex

Adapted from ***Pillay VV****. Pocketbook of Pesticide Poisoning for Physicians.* ***2001****. CBS Publishers, New Delhi.*

Treatment

An important differentiating point from organophosphates is that oximes are generally not recommended, while atropine can be given. Especially in carbaryl poisoning, oxime therapy can lead to the production of a carbamylated oxime which may be a more potent acetylcholinesterase inhibitor than carbaryl itself.[9] With other carbam-

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ate insecticides (particularly aldicarb), oximes may be a useful adjunct to atropine therapy.[10] But controversy persists.

ORGANOCHLORINES

Organochlorine pesticides are one variety of chlorinated hydrocarbons. There are 4 distinct categories of these pesticides[11]:

1. *DDT and analogues* - e.g., DDT (dichlorodiphenyltrichloroethane), and methoxychlor.
2. *Benzene hexachloride group* - e.g., benzene hexachloride (BHC), and gamma-hexachlorocyclohexane (lindane).
3. *Cyclodienes and related compounds* - e.g., aldrin, dieldrin, endosulfan (thiodan), endrin, isobenzan, chlordane, chlordecone (kepone), heptachlor, mirex (dechlorane).
4. *Toxaphene and related compounds* - e.g., toxaphene.

Table 9.3 lists Indian brand names of organochlorine pesticides.

Physical Appearance

These compounds are available as dusting powders, wettable powders, emulsions, granules, and solutions.

Uses

1. Insecticide
2. Gamma benzene hexachloride is used as a scabicide (treatment of scabies), and a pediculocide (eradication of head lice). It is available as topical ointment, cream, or lotion. Some Indian brand names include Bexarid (Shalaks), Gab (Gufic), Gamaric (Euphonic), Scaboma (Glenmark), and Ultrascab (Perch).

Usual Fatal Dose

DDT, lindane: 15 to 30 gm
Aldrin, dieldrin, endrin: 2 to 6 gm

Table 9.3 Common Organochlorine Compounds

Generic Name	Brand Name
Aldrin	Agroaldrin, Alcrop, Alditon, Aldrex, Aldrin 30, Mildrin 30, Tarmahit 30
BHC	Agrobenz D10, Agro BHC, Gamazene, Gammexane, Haxaman, Hexidole Hilbleach 50WP, Kargo BHC, Premodole 10EC, Sevidol (with carbaryl), Solchlor, Sudarshan, Sulbenz 50, Sunbrand
Chlordane	Agrodane 20EC, Chlordane, Mitox 20EC, Sudarshan 5EC, Termex, VegFru Chlortox
DDT	DDT Sudarshan 50, Didinex 25EC, Ramdit, Ranodit, Soltax, Suldit 50, Sunbrand, Tafarol, Tafidex
Dicofol	Kelthane
Endosulfan	Agrosulfan, Endohit, Hexasulfan, Hildon, Thiodan
Heptachlor	Agrochlor D5, Agrodono, Heptachlor, Heptaf 50, Heptar, Heptox, VegFru Heptex
Lindane	Agrodane, Bexarid, Emscab, Gab, Gamaric, Gamascab, Lindane 20, Lindex, Linsuline, Lintaf, Rasayan Lindane, Scabex, Scaboma, Scarab, Starbrand Lindane, Ultrascab

Adapted from **Pillay VV.** *Pocketbook of Pesticide Poisoning for Physicians.* **2001.** CBS Publishers, New Delhi.

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Toxicity Rating

Dieldrin is placed in the **extremely toxic** category (LD50 : 1 to 50 mg/kg), while DDT, endosulfan, and lindane are considered **highly toxic** (LD50 : 51 to 500 mg/kg), as per the **Insecticide Rules, 1971.**

Toxicokinetics

All the organochlorines can be absorbed transdermally, orally, and by inhalation. Many of these compounds are metabolised slowly and persist in tissues (especially fat) for prolonged periods.

Mode of Action

DDT and analogues affect the sodium channel and sodium conductance across the neuronal membrane especially of the axon.[12] They also alter the metabolism of serotonin, norepinephrine, and acetylcholine. The cyclodienes and lindane appear to inhibit the GABA-mediated chloride channels in the CNS.[13]

Clinical Features

Acute Poisoning:

1. GIT: nausea, vomiting, hyperesthaesia or paresthaesia of the mouth and face.
2. CNS: headache, vertigo, myoclonus, rapid and dysrhythmic eye movements, mydriasis, weakness, agitation, confusion, and convulsions.
3. Other systems: fever, aspiration pneumonitis, renal failure.

Chronic Poisoning:

Long term exposure to some of these compounds results in cumulative toxicity with manifestations such as weight loss, tremor, weakness, ataxia, abnormal mental changes, oligospermia, and increased tendency to leukaemias, thrombocytopenic purpura, aplastic anaemia, and liver cancer.

Diagnosis

1. Abdominal radiograph may reveal the presence of certain organochlorines which are radiopaque.
2. Organochlorines can be detected in serum, adipose tissue, and urine by gas chromatography.

Treatment

1. Decontamination – the same measures as detailed under organophosphate poisoning must be undertaken.
2. Seizures should be controlled with benzodiazepines, phenytoin, or phenobarbitone in the usual way.
3. Cholestyramine, a non-absorbable bile acid binding anion exchange resin is effective in enhancing the faecal excretion of organochlorine compounds.[14] It is administered at a dose of 16 gm/day for several days. It can be mixed with fruit juice and given orally (4gm, 6th hourly).
4. Hyperthermia should be managed aggressively with cooling.
5. Supportive measures – special attention must be paid to the airway and breathing, and adequate circulation should be maintained.
6. The following are contraindicated – oil-based cathartics, adrenaline, and atropine.

PYRETHRINS & PYRETHROIDS

Pyrethrins are active extracts of the **chrysanthemum** plant (*Chrysanthemum cinerariaefolium*), and include pyrethrum and piperonyl butoxide. Pyrethroids are synthetic analogues and number over 1000 varieties which are used as insecticides to incapacitate or "knock out" insects. Most mammals are resistant since they can rapidly metabolise and detoxify these agents. Common pyrethrins and pyrethroids available commercially in India are mentioned in **Table 9.4.**

Uses

These compounds are used as insect repellents and insecticides in household sprays, mosquito coils, and mats. They are also used to prevent pest infestation in granaries, and in agriculture as pesti-

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cides. They are sold as liquids, sprays, dusts, powders, mats, and coils.

Usual Fatal Dose

Pyrethrum has an LD50 of over 1gm/kg. Most cases of toxicity are actually the result of allergic reactions.[15]

Mode of Action

Structurally, pyrethroids are of 2 types -
Type I pyrethroids do not contain a cyano group. E.g., permethrin.

Type II pyrethroids contain a cyano group. E.g., deltamethrin, cypermethrin, fenpropathrin, fenvalerate, etc.

Like DDT, pyrethroids prolong the inactivation of the sodium channel by binding to it in the open state. Type II agents are more potent in this regard, and also act by inhibiting GABA-mediated inhibitory chloride channels.

Clinical Features

1. *Skin contact:* dermatitis, blistering.
2. *Eye contact:* irritation.
3. *Inhalation*: rhinorrhoea, sore throat, wheezing, dyspnoea.
4. *Ingestion* (large doses): paraesthesias, nausea, vomiting, vertigo, fasciculations, hyperthermia, altered mental status, seizures, pulmonary oedema, coma.

Table 9.4 Pyrethrins and Pyrethroids

Generic Name	Brand Name
Allethrin	Baygon mats, Pynamin Forte
D-allethrin	Baygon Knock-out aerosol, Baygon Power mats, Good Knight mats, Hit insect repellant
Cypermethrin	Agrocyper, Basathrin, Bilsif, Challenger, Cilcord, Cybil, Cymbush, Cymet, Cymperm, Cyper, Cyperguard, Cyperhit, Cyperin, Cypermethrin-Sandoz, Cypermil, Cypersul, Cyrux, Gilcyp Tech, Hilcyperin, Hypowder, Hycper, Motal, Parathrin, Ralathin, Ramceper, Ripcord, Shakti, Sicerin, Starcyprin, Superkiller, Tackle, Vegfrucott, Ustad
Decamethrin	Decathrin
Deltamethrin	Decis, Hexit (with allethrin), K-Othrine
Fenvalerate	Agrofen, Capvalerate, Fencidin, Fenhit, Fenkill, Fenval, Fighter, Gilfen, Parafen, Starfen, Sumicidin, Sumitox, Trumpcard
Fluvalinate	Marvik
Permethrin	Ambush, Lee, Permasect, Permethrin, Pounce
Pyrethrum	Tortoise mosquito coil

Adapted from ***Pillay VV.*** *Pocketbook of Pesticide Poisoning for Physicians.* ***2001.*** *CBS Publishers, New Delhi.*

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Diagnosis

1. Blood cholinesterase levels are normal.
2. ECG may demonstrate ST-T changes, sinus tachycardia, and ventricular premature beats.

Treatment

1. *Skin contact* - decontaminate with soap and water.
2. *Eye contact* - irrigate with normal saline or water for 10 to 15 minutes.
3. *Systemic poisoning* –
   a) Allergic reactions can be treated with epinephrine and antihistamines.
   b) In massive ingestions, stomach wash can be done after making sure that there are no petroleum distillate additives.
   c) Activated charcoal is beneficial.
   d) Oils and fats (including milk) promote the intestinal absorption of pyrethroids and should be avoided.
   e) Oxygen and ventilatory asistance must be administered as indicated.
   f) Bronchospasm is treated with standard bronchodilators.
   g) Seizures can be controlled with diazepam.
   h) Atropine and oximes are contraindicated, but some investigators recommend the former for drying up secretions.

Forensic Significance (For all insecticides)

Acute insecticide poisoning is a serious global problem accounting for an estimated 3 million cases of severe poisoning worldwide each year, with approximately 220,000 deaths. More than 90% of these cases are reported from developing countries such as India. In the UK, pesticides are responsible for only about 1% of deaths from poisoning while various studies in India indicate that the figures range from 20% to a staggering 70%![16,17] In the paediatric age group domestic pesticides can account for a substantial number of poisoning cases. Among the various pesticides, organophosphates and carbamates account for most of the reported cases of poisoning, though aluminium phosphide is making relentless inroads, especially in Northern and Central India.

Since most of the insecticides are easily available and relatively cheap, suicidal cases involving deliberate ingestion are quite common. A few homicides have also been reported.

STRYCHNINE

Strychnine is an alkaloid present in the seeds of *Strychnos nux vomica*, a tree belonging to family Loganiaceae. It is sometimes used as a rodenticide, and also for killing stray dogs.

Characteristics

Seeds are disc-shaped, greyish-brown, about an inch in diameter, and 1/4th inch in thickness. They are slightly concave on one side and convex on the other. The pericarp is very tough. Seeds are odourless but extremely bitter to taste.

Active Principles

Seeds contain

- *Strychnine:* odourless, colourless crystals, with bitter taste.
- *Brucine*
- *Loganine*

Mode of Action

Competitive antagonism of inhibitory neurotransmitter glycine at the post-synaptic spinal cord motor neurone.[19]

Clinical Features

- Anxiety, apprehension, nausea.
- Muscle twitching, convulsions, opisthotonus (spasmodic hyper-extension of spine), trismus (lock-jaw), risus sardonicus (spasm of facial muscles producing a characteristic grimace).
- Mydriasis.

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- Complications: hyperthermia, rhabdomyol-ysis, myoglobinuria, renal failure, respiratory failure.
- Consciousness is retained till the very end, resulting in an agonising death.

Differential Diagnosis

The most important condition with which it is often confused is tetanus. **Table 9.5** presents salient differences.

Treatment

- Diazepam or barbiturates for convulsions. If they are ineffective, neuromuscular blockade may have to be considered (after hooking the patient to a ventilator).[20]
- Oxygen, assisted ventilation.
- Decontamination measures (stomach wash) can be considered after convulsions have been controlled. Activated charcoal is beneficial.
- Acidification of urine can enhance the excretion of strychnine, but must be undertaken cautiously owing to the danger of exacerbating renal manifestations and metabolic acidosis.
- Treatment must be carried out in quiet and dimly-lit environment.

Usual Fatal Dose

5 to 8 mg/kg of strychnine ; 1 to 2 seeds. Seeds must be chewed or crushed before swallowing to be digested.

Autopsy Features

- Post-mortem caloricity.
- Early onset and disappearance of rigor mortis.
- Asphyxial signs.
- Congestion of g.i.tract.

In addition to the routine viscera, the following must be preserved for chemical analysis: spinal cord, one half of brain, and entire heart.

Forensic Significance

- Most cases of poisoning result from accidental ingestion of seeds (children), or "folk medicines" containing strychnine (adults).
- Owing to the agonising nature of manifestations, suicidal cases are rare.
- Homicidal cases have been reported, even though strychnine suffers from the major disadvantage of an extremely bitter taste which is difficult to mask. A landmark case from the 19th century is detailed in **Box 9.1**.

Table 9.5 Differences between tetanus and strychnine poisoning

	Tetanus	Strychnine Poisoning
History	Of injury	Of poisoning
Onset	Gradual	Sudden
Trismus	Early feature	Late feature
Involvement of muscles	Groups of muscles affected at a time	All muscles affected simultaneously
Muscle rigidity	Sustained even between convulsions	Disappears between convulsions
Chemical analysis	Negative	Positive for the poison

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Box 9.1

The Amazing Dr.Palmer

William Palmer was one of the worst poisoners the world has ever known, and his conduct is all the more heinous when one considers the fact that he was a qualified physician. Palmer graduated with a medical degree from St.Bartholomew's Hospital, London, in 1847, and married Annie Thornton the pretty (but illegitimate) daughter of a wealthy Army colonel. Annie and her bad tempered, drunken mother had inherited 20,000 pounds, which is probably why Palmer married her. Soon after the marriage, the mother died from "apoplexy" which was certified by Dr.Bamford, a kindly old man of over eighty with no qualifications but a high reputation. He completely trusted his young colleague, a fact which Palmer found very useful during the next few (busy) years. However the death of his mother-in-law did not improve Palmer's financial position because her money was tied up in certificates.

The following year, Palmer lost a lot of money at the races and had to borrow 600 pounds from a friend named Bladen. The latter died very soon thereafter, and Bamford helpfully signed the death certificate. Palmer managed to convince Mrs.Bladen that her husband had actually died owing him money ! This ploy was so successful that Palmer duplicated it later in the case of a young man named Bly. Bamford was at hand again to sign Bly's death certificate.

Next on Palmer's death list was his Uncle Joseph who was a drunkard. Since Palmer was scheduled to inherit his money upon his death, the good doctor decided his uncle was better off dead. He invited him to a brandy-drinking competition, which Joe accepted, drank whatever he was given, won hands down, vomited, and died.

Palmer next invited a wealthy old aunt to stay with him. To cure a slight indisposition, her affectionate nephew gave her a box of pills. But the ungrateful aunt thew them out of her window. It may or may not have been a coincidence, but the next morning, all Palmer's poultry were dead. The auntie had enough and quickly went home (presumably skipping breakfast).

By the end of 1853, Palmer was in dire financial straits owing to his compulsive gambling, and decided to insure his wife's life. He had an intuition of her impending demise. Very soon Annie did fall seriously ill and died. The death certificate mentioned cholera as the cause. Palmer received 13000 pounds from the insurance company. But most of this substantial sum went to his moneylenders. So Palmer had to get busy with the next victim on his list – his brother Walter. He insured the poor fellow's life for 14000 pounds. Within 6 months, Walter was dead. During the last days of his life, he was solicitously cared for by his loving brother who administered "costly medicines" unselfishly. The results of Walter's death were however not very satisfactory financially for Palmer. The insurance company delayed payment, and a demand to Walter's widow that she should forthwith settle a (non-existent) debt owed by his brother was rudely ignored.

Within a few weeks of Walter's unprofitable death, Palmer had once again run up huge debts and was in desperate need of money. On November 18, 1855, he marked out his next victim when he went to the races with his friends. It was John Parsons Cook who won a lot of money that day. The next day

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Palmer went to the hotel where Cook was staying, and hosted a party. He added antimony as well as strychnine to Cook's drinks who became violently ill. The following morning, Palmer looked in and must have been disappointed to find that Cook was still alive. But he concealed his disappointment under a brave smile, quietly slipped in some strychnine into his friend's coffee and left the hotel. An hour later, Cook was screaming and beating the bed in agony. However he survived through the day, and the next day was still alive though terribly weak. Nettled by Cook's selfish refusal to die, Palmer brought with him that morning, not only strychnine but also opium solution and prussic acid (cyanide). This was just too much for poor Cook and he died a few hours later. Palmer arranged for a death certificate to be issued immediately, but Cook's step father, a shrewd old man named Stevens insisted on a postmortem examination.

At 10 a.m on November 26, 1855, a few days after the death, Cook's body was autopsied. Dr.Harland, a barely competent pathologist without much experience was placed in charge. Instead of the privacy that is necessary for such a procedure, the autopsy was attended by no less than a dozen people which included also Dr.Palmer himself ! When the body was opened and the stomach was taken out, Palmer managed to spill most of the precious contents into the drain. Not satisfied with that, he even tried to subsequently tamper with the jar in which the stomach was placed, and went to the extent of offering a bribe to the cabman (who was deputed to take it to the laboratory) to break the container ! The cabman refused, and the jar eventually reached Professor Taylor, pathologist at Guy's Hospital, London, for analysis. It was hardly surprising however, that his report was negative for strychnine or for that matter, any poison.

A second autopsy was then undertaken and other organs were submitted for analysis, which revealed substantial quantities of strychnine. Just before the inquest, Palmer tried desperately to bribe the coroner by offering him money, meat, and fish ! Nothing worked, and finally Palmer was committed to trial on a charge of murder. The trial lasted for 14 days, and Palmer was convicted and sentenced to death. On the morning of June 14, 1856, just before his execution, Palmer was offered (as is customary), a glass of wine. He accepted it and drank slowly, saying "I always get indigestion the next morning if I drink in a hurry !"

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CHAPTER - 10

Deliriants and Psychotropics

DELIRIANTS

Deliriant drugs act on the brain and induce delirium, i.e., altered consciousness with confusion, delusions, hallucinations, and agitation. Important examples include **datura, cannabis,** and **cocaine.** Actually the last mentioned should more rightly be classified as a stimulant, but it is conventional (even if not accurate), to include it among the deliriants.

DATURA

Common Name
Jimson weed, thorn apple, stinkweed.

Family
Solanaceae

Characteristics
Indian species include *Datura fastuosa, D. atrox,* and *D. metel.* There are two varieties of D.fastuosa – niger (with purple flowers), and alba (with white flowers). Leaves are dark green with pointed margins, and the flowers are tubular. The fruit capsule has spines (**Fig 10.1**), and contains numerous reniform seeds. They bear a superficial resemblance to chilly seeds, but are very bitter to taste. **Table 5.1** (page no. 342), mentions important differentiating points.

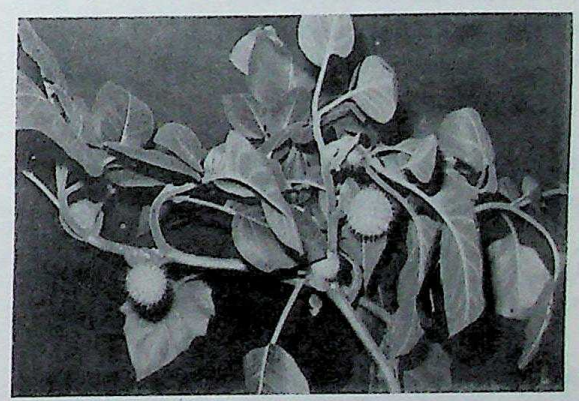

Fig 10.1 Datura

Active Principles
Seed is the most toxic constituent and contains the following active principles :

1. *Hyoscine* (*scopolamine*)
2. *Hyoscyamine*
3. *Atropine*

Uses
One or the other active principle is used as

- Mydriatic
- Antispasmodic
- Antidote for organophosphates & carbamates

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- Pre-anaesthetic medication
- Bronchial asthma (now obsolete)

Clinical Features

Summarised in the classic phrase - *blind as a bat, hot as a hare, dry as a bone, red as a beet, and mad as a wet hen.*

Main features can be better remembered as a series of D's:

- Dryness of mouth, nausea, vomiting
- Dysphagia
- Dilated pupils
- Diplopia
- Dry, hot skin
- Drunken gait (ataxia)
- Delirium, with confusion, agitation, and hallucinations
- Drowsiness leading to coma
- Dysuria (urinary retention)
- Death due to respiratory failure or cardiac arrhythmias

As would be evident from the above, the clinical picture is one of **anticholinergic poisoning.**[1].

Usual Fatal Dose

50 to 100 seeds

Autopsy Features

Non-specific findings.

Forensic Significance

1. *Accidental*:
   - Therapeutic misadventure due to quackery.
   - Ingestion of seeds by children.
2. *Suicidal*: Mostly reported from rural areas.
3. *Homicidal*: Extremely rare.
4. *Stupefaction*: When an extract of the seeds is mixed with food (or sometimes tobacco) and administered to an unsuspecting victim, it produces confusion, agitation, disorientation, and loss of consciousness, thereby facilitating robbery or rape. Cases have been reported of gullible travellers being "stupefied" by copassengers with eatables or cigarettes containing datura extract, and then relieved of their valuables.

CANNABIS

Common Name

Indian hemp

Family

Cannabinaceae

Characteristics

The term **cannabis** refers to the flowering and fruiting tops of *Cannabis sativa*, which is a tall weed growing up to 15 feet in height. It is a dioecious plant, i.e., the sexes are separate.

Active Principle

Δ^1 tetrahydrocannabinol, (i.e., Delta[1] tetrahydrocannabinol)

Common preparations

1. **Marijuana** (marihuana, mary jane, pot, weed, grass): dried flowering tops crushed and smoked in a pipe, or in the form of a cigarette (reefer or joint).
2. **Hashish** (charas): dried resin of flowering tops.
3. **Ganja:** resinous extract of leaves and bracts of female plant.
4. **Bhang:** dried leaves and stem made into a decoction.
5. **Majun:** sweetmeat prepared with plant extract.
6. **Sinsemilla:** seedless marijuana (unpollinated female plant).

There is a characteristc odour to all cannabis preparations, especially to the smokable forms, described as being akin to that of a burnt rope.

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Uses

1. Antiemetic against nausea and vomiting induced by anicancer drugs.
2. Possible role in the treatment of convulsions, anxiety, pain, and inflammation.

At present, cannabis is a banned drug in India and most parts of the world.

Clinical Features

Acute Poisoning: inhalation is associated with more pronounced effects than ingestion.

- Euphoria with drowsiness and disorientation.
- Motor incoordination.
- Stimulation of appetite.
- Tachycardia.
- Conjunctival congestion, miosis.
- Large doses produce nausea, anxiety, confusion, delusions, and hallucinations.
- Characteristic (burnt rope) odour, if the drug has been used for smoking.
- Intravenous use can cause headache, diplopia, vertigo, dyspnoea, hypotension, and renal failure.

Chronic Poisoning:

- **Amotivational syndrome:** Chronic abusers of cannabis gradually become lethargic, apathetic, and disinclined to work, with poor concentration.
- **Hashish insanity:** Chronic, heavy abuse of cannabis causes paranoid psychosis with violent behaviour, culminating in homicide or suicide (*running amok*).[2]
- Increased susceptibility to pharyngitis, bronchitis, asthma, and gynaecomastia (in males).

Treatment

Acute Poisoning:

- Decontamination - stomach wash in case the drug has been ingested.
- Haloperidol or other antipsychotic medication for psychosis.
- Psychotherapy.

Chronic Poisoning:

- Gradual withdrawal of the drug.
- Diazepam for anxiety.
- Antipsychotics for psychosis.
- Psychotherapy.

Forensic Significance

Cannabis is a drug of abuse, being particularly popular among sanyasis and sadhus, western musicians and painters, and college going youth. Long term effects are generally not serious, and physical dependence is uncommon. However abrupt stoppage after habitual use can result in a mild withdrawal reaction characterised by restlessness, insomnia, anorexia, and nausea.

COCAINE

Common Names

Coke, snow, cadillac, white lady, etc.

Family

Erythroxylaceae.

Characteristics

Cocaine is an alkaloid obtained from the leaves of the coca tree, of which there are two important species: *Erythroxylon coca* and *E. novogranatense*. They grow well in South America, Indonesia, and India. Today however cocaine can be synthesised in the laboratory, and is often sold as **crack**, which is a smokable form obtained by combining cocaine hydrochloride with baking soda and water, followed by heating and drying.

Cocaine occurs as white, shiny crystals with a bitter, numbing taste.

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Uses

1. Topical anaesthetic for cornea and ENT procedures (formerly).
2. **Brompton's cocktail :** Cocaine is part of this mixture which was previously popular as a pain reliever in terminal cancer.[3] The other constituents include morphine, chlorpromazine, and alcohol.

Clinical Features

Acute Poisoning[4]

Cocaine is usually abused by nasal insufflation (snorting or sniffing), but it can also be smoked, ingested, or injected intravenously.

1. **Stage of early stimulation:** Euphoria, excitement, mydriasis, tachycardia, hypertension, tachypnoea, vertigo, nausea.
2. **Stage of advanced stimulation:** Vomiting, muscle twitching, convulsions, dyspnoea, hyperthermia (*cocaine fever*), hallucinations, circulatory and respiratory failure.
3. **Stage of depression:** Paralysis of muscles, loss of reflexes, coma and death.

Bodypacker syndrome (Minipacker syndrome, Bodystuffing) - Cocaine is sometimes smuggled across international borders by individuals (bodypackers, bodystuffers, or mules), who swallow several small plastic bags or balloons or even condoms filled with cocaine and then take a flight into another country. On arrival at their destination, they check into their hideout, self-administer cathartics, and defecate the ingested packets of cocaine. Sometimes rectal suppositories or disposable enemas are used. The packets are retrieved, cleaned, and sold illegally. Sometimes however, the packets may burst while in transit through the digestive tract releasing massive quantities of cocaine, leading to overwhelming overdose and collapse.

Although this is more commonly associated with smuggling of cocaine, it has also been reported in the case of other drugs, especially heroin.[5] Diagnosis of an asymptomatic body packer can be accomplished with the help of an abdominal x-ray, ultrasound, or CT scan. The last two methods should be resorted to only if x-ray is unclear. In some cases, faecal examination over a period of a few days may be necessary.[6]

Chronic Poisoning

Addiction leads to tolerance. Cocaine is usually abused by the upper classes of society to enhance self-image or improve professional performance. There is a tendency for compulsive use of the drug though classical physical dependence is uncommon. While "snorting" or "sniffing" is still the favoured manner of intake, smoking **crack** (free-based cocaine) is becoming increasingly popular. Immediately upon intake, there is euphoria (*rush*), followed about an hour or so later by rebound depression (*crash*). To rid himself of the unpleasant effects of the latter, the individual feels compelled to take the drug again, and the vicious cycle continues until physical, finacial, or drug resources are exhausted.

Features:

- Anorexia, emaciation.
- Hyperactivity, flight of ideas.
- Insomnia.
- Mydriasis.
- Tachycardia, hypertension.
- Tachypnoea.
- Psychosis. Tactile hallucinations are common: feeling of insects crawling on the skin, or of sand lying under the skin (**coke bug, formication,** or **Magnan's symptom**).
- Complications:
  Nasal insufflation - rhinitis, nasal erosions or perforation, sinusitis.
  Smoking - chronic cough, bronchitis.

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IV abuse - hepatitis, AIDS, venereal diseases, endocarditis, osteomyelitis, etc.

Treatment

Acute Poisoning

1. Decontamination (stomach wash, activated charcoal, etc.), if the drug has been ingested.
2. Benzodiazepines or barbiturates for convulsions.
3. Propranolol for hypertension and cardiac arrhythmias.
4. Haloperidol for psychosis.
5. Ice bath for hyperthermia.
6. Supportive measures.

For Bodypacker syndrome

- Asymptomatic patients should be treated by whole bowel irrigation with polythylene glycol solution. However, some investigators do not approve of this method since the polythylene glycol can dissolve the heroin from a package, rupturing it and increasing absorption of heroin. Instead, a period of waiting is recommended so that all packages pass into the colon from the stomach, and then low-volume phosphosoda enemas or high-volume saline enemas are administered. Food ingestion must not be permitted until all packages have moved into the colon. Metoclopramide 10 mg, 8th hourly, may be administered to encourage gastric emptying. Bowel obstruction must be ruled out. It may be advisable to empty the rectum first by a bisacodyl suppository.

- Symptomatic patients must be managed with specific drugs, activated charcoal, and whole bowel irrigation. Intestinal perforation or obstruction by packets requires surgical intervention.

Chronic Poisoning

1. Gradual withdrawal of the drug.
2. Psychotherapy.

Usual Fatal Dose

1 - 1.2 gm orally or intranasally.

Autopsy Features

- Evidence of nasal erosions, ulceration, or perforation (in "snorters"). Nasal swabs must be taken for chemical analysis.
- Cerebral and pulmonary oedema.
- Generalised visceral congestion.

Forensic Significance

1. Drug of abuse.
2. Accidental overdose sometimes leads to death.

PSYCHOTROPICS

Psychotropic drugs are substances that affect psychic function, behaviour, or experience. There are 3 classes:

1. *Antidepressants* - E.g., amphetamine, tricyclic antidepressants, monoamine oxidase inhibitors.
2. *Neuroleptics* - Also referred to as antipsychotics or major tranquillisers. E.g., phenothiazines, butyrophenones, indoles, etc.
3. *Hallucinogens* - Also referred to as psychedelics. E.g., LSD, phencyclidine, psilocybine, etc.

Only **amphetamine, LSD,** and **phencyclidine** will be discussed here.

AMPHETAMINE

Amphetamine was first synthesised in 1887, but began to be therapeutically used only since the 1930s. Because of its abuse potential, its therapeutic administration is greatly restricted today. Chemically, amphetamine is phenylpropanolamine.

Derivatives

Methamphetamine, dextroamphetamine, fenfluramine, phentermine, mephentermine, methyl pheni-

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date, and synthetic amphetamines, i.e., **designer drugs** - methylenedioxyamphetamine (MDA or Love Drug), methylenedioxymethamphetamine (MDMA or Ecstacy), and methylenedioxyethamphetamine (MDEA or Eve).

Uses

- Narcolepsy.
- Attention deficit disorder.
- Hypotension (mephentermine as i.v solution).

Mode of Action

Amphetamines enhance the synaptic concentration of dopamine and norepinephrine, either by direct release from storage vesicles or by inhibition of re-uptake. They are powerful stimulants of the CNS and CVS.

Clinical Features

Acute Poisoning

Mild: Restlessness, insomnia, tremor, sweating, dry mucous membranes, nausea, mydriasis.

Moderate: Hyperactivity, confusion, hypertension, tachycardia, tachypnoea, vomiting, hallucinations.

Severe: Delirium, hyperpyrexia, convulsions, arrhythmias.

Chronic Poisoning

1. *Amphetamine psychosis* :
   - Stereotypy – obsessed with repetitive activities involving grooming, rearranging, cleaning, etc.
   - Paranoid behaviour – characterised by suspicion, hostility, and anxiety.
   - Delusions – of persecution.
   - Hallucinations – usually visual.
2. *Gilles de la tourette syndrome* : characterised by tics, eye blinking, jaw jerks, humming, etc.
3. *Cardiomyopathy*
4. *Intracranial haemorrhages*

Treatment

Acute Poisoning

- Decontamination.
- Acidic diuresis (controversial).
- Benzodiazepines or antipsychotics for agitation.
- Sodium nitroprusside, i.v, for hypertension.
- Ice bath or cold sponging for hyperthermia.
- Benzodiazepines or phenytoin for convulsions.
- Supportive measures.

Chronic Poisoning

- Chlorpromazine or haloperidol for amphetamine psychosis.
- Haloperidol for Gilles de la tourette syndrome.

Usual Fatal Dose

250 mg (addicts can tolerate much larger doses).

Forensic Significance

- Acute overdosage with amphetamines is uncommon in India.
- Formerly, amphetamines were widely abused by those who were compelled to stay awake at nights periodically, e.g., truck drivers, night shift industrial workers, medical students, etc.
- Intermittent amphetamine abuse is characteristically seen in athletes who wish to enhance their endurance and performance.
- Today "designer drugs" are abused by youngsters during the course of *rave parties*, for dancing all night long.

LYSERGIC ACID DIETHYLAMIDE (LSD)

Common Names

Acid, microdot, purple haze, white lightning, etc.

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Physical Appearance
Crystalline substance which is colourless, odourless, tasteless, and water-soluble. It is effective in extremely small doses (micrograms).

Sources
1. Ergot
2. *Rivea corymbosa* (a Mexican plant) of family Convolvulaceae.
3. Morning Glory plant (contains a compound very similar to LSD).

Clinical Features
- Mydriasis, tachycardia, tremor, ataxia.
- Hallucinations.
- Panic attacks, psychotic reactions, flashback phenomena, mania.
- Convulsions, focal cerebral deficits, coma.
- Hyperthermia, bleeding disorders.

Treatment
- Antipsychotics.
- Benzodiazepines.
- Quiet, secluded environment
- Supportive measures.
- Psychiatric follow-up.

Forensic Significance
LSD is the most powerful hallucinogen known to man. It was popular among the hippies in the West in the 1960s. Suicidal and accidental deaths arising out of bizarre behaviour induced by the drug have been reported. Repeated use by an addict can lead to permanent psychosis.

PHENCYCLIDINE

Common Names
Angel dust, PCP, peace pill, hog, rocket fuel, cadillac, super grass, etc.

Physical Appearance
White crystalline powder.

Clinical Features [8]
Phencyclidine is usually smoked, sniffed, or injected (iv, sc).
- Nystagmus, miosis, ataxia, tremor, dysarthria, tachycardia, hypertension.
- Lethargy, catatonia, coma.
- Acute psychosis with delusions and hallucinations.
- Agitation, violent tendency, bizarre behaviour.

Treatment
- Decontamination is usually not applicable, since the drug is rarely taken orally.
- Acid diuresis.
- Supportive care.
- Psychiatric follow-up.

Forensic Significance
Phencyclidine is a popular drug of abuse in the West. A common mode of intake involves sprinkling the drug on parsley or marijuana leaves, and then smoking it. It is sometimes used as an adulterant in expensive drugs of abuse such as cocaine. Addiction leads to violent tendency, psychosis, and suicidal as well as homicidal behaviour.

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CHAPTER - 11

Cardiotoxic Poisons

Examples of poisons which act directly on the heart include **oleander** (Nerium and Thevetia), **aconite, digitalis, nicotine, quinine,** and **quinidine.**

NERIUM

Common Names
Common oleander, pink oleander, rose laurel

Family
Apocynaceae

Characteristics
Nerium oleander is the European species, while *N.odorum* is commonly seen in India, (**Fig 11.1**). It is a large ornamental shrub commonly seen in gardens. Leaves are long and pointed (lanceolate), while the flowers are white or pink, and are arranged in clusters.

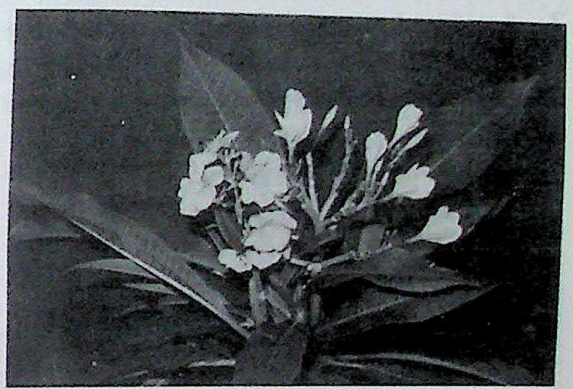

Fig 11.1 Nerium Odorum

Active Principles
All parts of the plant are toxic, particularly the stem, seeds, and the root. Important active principles include

- *Oleandrin*
- *Neriin*
- *Folinerin*
- *Rosagenin*

Uses
1. Various parts of the plant find use in folk remedies for various ailments.
2. Illicit abortifacient.

Clinical Features
- Nausea, vomiting.
- Ventricular tachycardia, fibrillation, AV block. Death results from cardiac failure.

Usual Fatal Dose
- 5 to 15 leaves.
- 15 gm of root.

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Treatment

- Decontamination in the usual way (stomach wash, cathartics).
- ECG monitoring.
- Correction of cardiac arrhythmias with antiarrhythmic drugs such as lidocaine and phenytoin. AV block often responds to atropine.
- Correction of fluid and electrolyte disturbances

Forensic Significance

1. Because of the easy availability of this poison, it is commonly encountered as a suicidal agent, particularly in rural areas. For this purpose, usually a decoction of the root is prepared and consumed.
2. Accidental poisoning may result from its misuse as folk remedy.
3. The extract of this plant, or a twig or root may be employed to procure illicit abortion.

THEVETIA

Common Name

Yellow oleander, lucky nut

Family

Apocynaceae

Characteristics

It is a large ornamental shrub like Nerium, but the leaves are oblong, and the flowers are tubular and yellowish. Fruits are black, globular, and fleshy. The most common Indian species is *Cerbera thevetia*, (**Fig 11.2**). The most toxic parts of the plant are the leaves and the fruits.

Active Principles

- *Thevetin*
- *Thevetoxin*
- *Neriifolin*

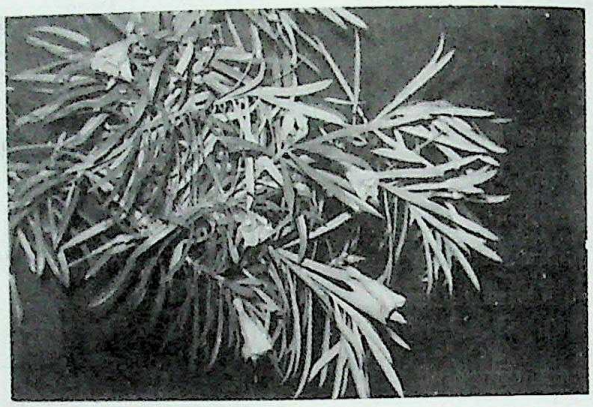

Fig 11.2 Cerbera Thevetia

Clinical Features

- Decontamination
- ECG monitoring
- Correction of cardiac arrhythmias, and fluid and electrolyte disturbances

Usual Fatal Dose

5 to 10 leaves, or 5 to 8 seeds.

Treatment

- Nausea, vomiting.
- Ventricular tachycardia, fibrillation, AV block. Death results from cardiac failure.

Forensic Significance

Owing to easy accessibility, suicidal poisoning with extracts of this plant is fairly common in rural areas of this country. A common method involves the consumption of a paste made out of the seeds.

A related species of plant *Cerbera odallum* (**Ordeal tree, Tangena nut**), is a small tree that grows

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well in South India, especially Kerala. It bears fruits which closely resemble unripe (green) mangoes. Accidental poisoning can result from mis-taken identity. The kernel of the fruit is extremely toxic, and contains the active principle *cerberin*. Manifestations are similar to those seen with oleander (*vide supra*). Suicidal ingestion of tangena nut is fairly common in Kerala.

ACONITE

Common Names
Monks hood, wolf bane

Family
Ranunculaceae

Characteristics
The term **aconite** refers to the root of the aconitum plant, of which there are several species. *Aconitum napellus* is the European species. Indian species are encountered in the sub-Himalayan region, and include *Aconitum ferox*, *A.chasmanthum*, and *A.balfouri*. The plant itself is a large herb, with palmate leaves (**Fig 11.3**), and white or blue flowers. The most toxic part is the root, which is bitter-sweet in taste, and bears a superficial resemblance to the edible horse radish.

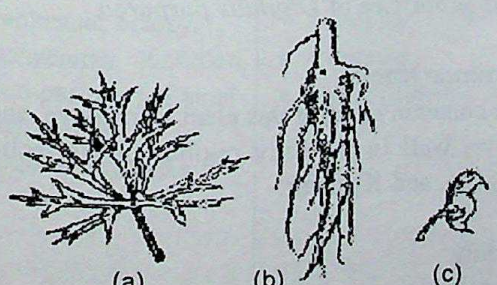

Fig 11.3 Aconite (a) Leaf (b) Root (c) Flower

Active Principles
Indian species :
- *Hypoaconitine*
- *Mesoaconitine*

European species:
- *Aconitine*
- *Mesaconitine*
- *Jesaconitine*

Uses
Aconite is used in various folk remedies, including Ayurveda.

Clinical Features
- Tingling and numbness of mouth and throat.
- Nausea, salivation, vomiting.
- Muscle weakness, convulsions.
- Dilated pupils. Sometimes hippus is seen, i.e., a spasmodic pupil.
- Hypotension, cardiac arrhythmias, AV block.

Usual Fatal Dose
Root: 1gm
Aconitine: 3 to 5mg

Treatment
1. Decontamination : emesis, stomach wash.
2. Cardiac monitoring in intensive care unit.
3. Treatment of cardiac toxicity with lidocaine, procainamide, or phenytoin.

Forensic Significance
1. Aconite is often touted as an "ideal homicidal poison" (**Table 11.1**). but its actual employment in homicides is quite rare. A landmark case is mentioned in **Box 11.1**.

2. Accidental poisoning can arise from mistaken identity with horse radish, which is edible. To differentiate the two, the cut surface must be examined. A pinkish tinge develops gradually in the case of aconite.

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3. Suicidal ingestion of the root is not uncommon in those areas where the plant grows well.

4. Illicit abortifacient.

Table 11.1 Aconite as an "Ideal" homicidal poison

- Easily available.
- Inexpensive.
- Kills in very small doses.
- Can be easily mixed with food or drink without arousing suspicion.
- Symptoms resemble natural disease (heart disease).
- Difficult to detect by chemical analysis.

Box 11.1

Dr.Pritchard: Pathological Liar and Poisoner

Dr.Pritchard born in 1825, was the son of a Captain in the Royal Navy. After obtaining his medical degree, he served as an assistant surgeon in the Navy, before marrying Mary Jane Taylor in 1851 and settling down in Yorkshire as a general practitioner. But Dr.Pritchard was a compulsive liar and a sexual profligate, and soon lost his reputation with the townspeople. He moved to Glasgow, where too his deviant behaviour got him into trouble.

In early 1864, Dr.Pritchard had an affair with his maid servant Mary M'Leod, which was discovered by his wife, and led to bitter quarrels. In November 1864, Mrs.Pritchard fell ill with vomiting, abdominal pain, and generalised weakness. She also developed cardiac problems. Her concerned mother, Mrs.Taylor arrived to look after her. However, within a few days she too fell ill. Dr.Pritchard who had been administering an overdose of tincture of aconite to his wife in an attempt to kill her, had decided to combine aconite with antimony for her mother. Mrs. Taylor died on 24 February, 1865. Her death was certified as "apoplexy" by Dr.Pritchard himself.

After his mother-in-law's death, Dr.Pritchard reverted his attention back to his wife and resumed poisoning her with aconite. Periodic poisoning finally led to her death on 17 March, 1865. Dr.Pritchard certified her death as "gastric fever", and took her body to Edinburgh for burial. However, on his return to Glasgow, he was arrested by the police on suspicion of murder. The bodies of Mrs.Taylor and Mrs.Pritchard were exhumed. Antimony was found in both bodies. Aconite being extremely difficult to detect (especially in those days when methods of analysis were primitive), was not found in either victim. Dr.Pritchard was charged with both murders, tried and convicted.

On the night before his execution, he confessed to both murders providing details of administration of antimony and aconite (mostly mixed with foodstuffs or drink). His motive for the murders remains unclear to this day, though it appears to be related to marital discord arising out of his affair with Mary M'Leod.

DIGITALIS

The history of cardiac glycosides in relation to the treatment of congestive heart failure begins with the classic **1785** monograph by **William Withering**, who wrote about the therapeutic and toxic properties of *Digitalis purpurea.*

Common Name

The common name of this plant is foxglove, and it grows well in the hilly regions of Darjeeling, Nilgiris, and Kashmir.

Family

Scrophulariaceae

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Characteristics

It is a biennial or perennial herb, growing upto 1 to 1.5 metres in height. Leaves are hairy, ovate, toothed, and gray-green in colour, while the flowers are tubular and pink or white in colour. There is a related species, *Digitalis lanata*, which is also rich in cardiac glycosides. Leaves constitute the main source of glycosides in both plants.

Active Principles

- *Digoxin*
- *Digitoxin*

Toxicokinetics

Both digoxin and digitoxin are well absorbed orally, but while the former is only moderately protein bound and has a large apparent volume of distribution (adults : 7 to 8 L/kg, neonates : 10 L/kg, infants : 16 L/kg), digitoxin is highly protein bound and has a low apparent volume of distribution. 60 to 80% of digoxin is excreted unchanged in the urine and the terminal half-life is about 36 hours. Digitoxin has a much longer elimination half-life (about 100 hours).

Most of an administered dose of digoxin is distributed to skeletal muscle after absorption (about 65%). The myocardium:plasma ratio is about 30:1.

Mode of Acion

Digitalis glycosides inhibit active transport of Na^+ and K^+ across cell membranes by binding onto a specific site on the extracytoplasmic face of the a subunit of Na^+-K^+-ATPase. The force of contraction of the heart (positive inotropic effect) is increased due to increase in cytosolic Ca^{++} during systole. Both Na^+ and Ca^{++} enter the myocardial cells during each cycle of depolarisation, contraction, and repolarisation. During repolarisation and relaxation, Ca^{++} is pumped back into the sarcoplasmic reticulum by Ca^{++}-ATPase and is removed intracelllarly by a Na^+Ca^{++} exchanger, and a sarcolemmal Ca^{++}-ATPase[2].

Clinical Features

Manifestations of digitalis overdose are mentioned separately for adults and children in **Table 11.2**.

Poor prognosis is associated with old age, plasma digoxin level exceeding 15 ng/ml, and presence

Table 11.2 Acute Digitalis Poisoning

Adults		Children	
Cardiac	Non-Cardiac	Cardiac	Non-Cardiac
▪ AV block (high degree) ▪ Ventricular ectopy, tachycardia, fibrillation ▪ Non-paroxysmal atrial tachycardia ▪ Sinus bradycardia	▪ Anorexia, nausea, vomiting ▪ Confusion ▪ Hyperkalaemia	Often not severe ▪ Bradycardia, ▪ AV block (I or II degree) ▪ ST segment depression ▪ Junctional rhythm In severe cases ▪ AV junctional tachycardia alternating with slow ventricular rate ▪ Ventricular fibrillation	▪ Vomiting ▪ Lethargy

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of AV block (high degree), hyperkalaemia, or ventricular tachycardia.

Usual Fatal Dose

Digitalis leaf: 2 gm.
Digoxin: 10 mg.
Digitoxin: 3 mg.

Therapeutic plasma concentration of digoxin should not exceed 2 ng/ml. Concentrations exceeding 15 ng/ml are potentially fatal.

Treatment

1. **Initial Treatment:**
   a) Decontamination: Emesis, lavage, activated charcoal, cathartic (as applicable).
   b) Forced diuresis, haemodialysis, and hamoperfusion are generally ineffective.

2. **Advanced Treatment :**
   a) **Antidote :** Digoxin-specific antibody fragments (Fab).

      Fab therapy is of proven efficacy in not only digitalis overdose, but also in red squill and oleander poisoning.[3] Fab fragments are administered intravenously. They bind intravascular free digoxin and then diffuse into the interstitial space and bind free digoxin there. Fab therapy has been reported to produce dramatic improvement in life threatening digitalis poisoning.[4]

      Dose: Use an empiric dose, based on average requirements for an acute or chronic overdose in an adult or child, as mentioned below:

      Adult = 10 to 20 vials (acute poisoning), 3 to 6 vials (chronic poisoning).

      Child = 10 to 20 vials (acute poisoning), ¼ to ½ vial (chronic poisoning).

      Each vial (Digibind) contains 38 mg of Fab fragments.

      Mode of Administration: Intravenous, over 30 minutes, via a 0.22 mm membrane filter. Reconstitute each vial with 4 ml of sterile water and use immediately (or within 4 hours if refrigerated).

      Adverse Effects: Allergic reactions (rare).

      Disadvantages: Not yet available in India. Can be procured from abroad, but is extremely expensive. A full course of antidotal therapy with Fab fragments can cost between 2000 to 3000 dollars !

   b) In the absence of Fab fragments, ventricular irritability can be treated with phenytoin or lidocaine.

      Dose: Phenytoin-50mg/min, slow i.v (maximum 1000 mg in adult, 15 to 20mg/kg in child). Maintenance oral dose:300 to 400mg/day in adult, 6 to 10mg/kg/day in child.
      Lidocaine – 1 mg/kg i.v bolus, followed by continuous infusion at 1 to 4 mg/min in adult, or 20 to 50 mg/kg/min in child. 15 minutes after initial bolus, an additional 1 mg/kg i.v bolus should be administered (both adult and child).

   c) In patients with severe supraventricular bradyarrhythmias, or high degrees of AV block, 0.5 mg of atropine is given i.v in an adult, (0.02 mg/kg in child, minimum being 0.1 mg). This dose can be repeated every 5 minutes as necessary.

   d) External or transvenous pacemaker.

   e) Treatment of hypo-/hyperkalaemia, and hypomagnesaemia, as follows -

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Hypokalaemia: i.v potassium chloride in 0.9 or 0.45 % sodium chloride, at a rate of 0.5 to 1.0 mEq/min (1 mEq/kg/hour in a child).

Hyperkalaemia : i.v insulin, dextrose, sodium bicarbonate, and oral ion-exchange resins (sodium polystyrene sulfonate). Do not administer calcium salts.

Hypermagnesaemia: 2 gm magnesium sulfate (10%) i.v over 20 minutes (25 mg/kg/ dose in a child). Maintenance : 1 to 2 gm/ hr (adult), 25 to 50 mg/kg/hr (child).

Forensic Significance

Digitalis poisoning (acute and chronic) is an important pharmacotoxicological problem. Acute toxicity may result from accidental ingestions (children), deliberate overdose (adults), or inadvertent dosing errors (elderly). Chronic toxicity is usually iatrogenic.[5]

QUININE and QUINIDINE

The bark of the South American **cinchona tree** yields both quinine and quinidine which have been around for more than 350 years. Quinidine is a more powerful antimalarial than quinine, but is unfortunately also much more toxic.[6]

Uses

1. Quinine is mainly indicated in the treatment of chloroquine-resistant and multidrug-resistant falciparum malaria.
2. Quinine is also reportedly beneficial for relieving nocturnal leg cramps, and for the symptomatic relief of myotonia congenita.
3. Quinidine is a potent antiarrhythmic agent and is used for the maintenance of sinus rhythm (in atrial flutter/fibrillation), as well as for the prevention of ventricular tachycardia/fibrillation.[7]

Toxicokinetics

Cinchona alkaloids are rapidly absorbed when given orally or intramuscularly, but the latter mode of administration often leads to tissue necrosis. Peak plasma levels are achieved in 3 hours. Both quinine and quinidine are highly protein-bound. The liver, kidneys, and muscles metabolise 80% of the ingested dose, while 20% is excreted unchanged in the urine. Renal excretion is greatly enhanced in acidic urine.

Clinical Features

Repeated therapeutic doses, as well as overdose result in a cluster of manifestations referred to as **Cinchonism** which is characterised by the following:

1. *Mild:* Tinnitus, headache, nausea, disturbed vision.
2. *Severe:* Vertigo, deafness, mydriasis, photophobia, diplopia, constricted visual fields, scotomata, blindness (quinine amblyopia), vomiting, diarrhoea, abdominal pain, sweating, skin rash, hypoglycaemia, and cardiac arrhythmias. Rapid infusion can cause severe hypotension.
3. *Potentially lethal:* delirium, coma, seizures, myocardial depression.

Quinine and quinidine are contraindicated in myasthenia gravis (can aggravate muscle weakness), and in pregnancy (can induce abortion due to oxytocic action). Special care is necessary in diabetics, since the hypoglycaemic effect of antidiabetic drugs can get aggravated.

Quinine can sometimes produce a hypersensitivity reaction characterised by massive haemolysis, haemogloinuria with passage of dark urine, anuria, and renal failure (black water fever). It occurs usually during the course of treatment of falciparum malaria.

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Usual Fatal Dose

Quinine: 4 to 8 gm.
Quinidine: 4 to 6 gm.

There are however reports of survival following ingestion of upto 20 gm of quinidine.[8]

Diagnosis

Measurement of serum levels of these drugs by high pressure liquid chromatography: Quinine levels higher than 15 mg/ml, and quinidine levels higher than 10 mg/ml are indicative of serious toxicity.

Early recognition of visual damage is essential. Eye changes (photophobia, misty vision) usually begin in ½ to 1 hour after overdose and may progress to partial or complete blindness in 6 to 12 hours. While such blindness may resolve over a period of time (1 to 3 weeks), it sometimes becomes permanent.

Treatment

1. Emesis is contraindicated. Stomach wash (with prior endotracheal intubation) should be undertaken only if the patient is seen within 1 to 2 hours of ingestion.

2. Activated charcoal has been proved to be beneficial and can be administered in the usual manner (1 gm/kg).[9,10]

3. While acid diuresis promotes clearance of quinine and quinidine, it is associated with cardiotoxicity and hence is not recommended. On the other hand, serum alkalinisation with sodium bicarbonate can be beneficial.[11] It is desirable to achieve and maintain a serum pH of 7.45 to 7.50.

4. Continuous cardiac monitoring is essential. Alkalinisation of serum usually prevents conduction abnormalities and arrhythmias. Administration of Class I, II, and III antiarrhythmic agents is contraindicated.[12] Ventricular tachycardia/fibrillation can be corrected by direct-current cardioversion. Torsade de pointes can be tackled with isoproterenol or overdrive pacing, while transvenous pacing is useful for complete heart block.

   Correction of acidosis, electrolyte imbalance, and hypoxia are imperative for the successful management of cardiotoxicity.[13]

5. Visual damage may be minimised by stellate ganglion block, but there appears to be some controversy regarding this.[14] Specific treatment for quinine retinal toxicity has so far not been evolved.[15] Vision usually improves with appropriate prompt reversal of systemic toxicity, though pupils may remain dilated for a long time even after complete restoration of eyesight.[16]

6. Haemodialysis combined with resin haemoperfusion may help in eliminating quinine from the blood.[17] Exchange transfusion does not appear to be beneficial.

NICOTINE

Nicotine is the active principle present in the leaves of the Nicotiana (**tobacco**) plant, of which there are two species : *Nicotiana tabacum* and *N. rustica.*

Family

Solanaceae

Characteristics

It is an ornamental shrub with inconspicuous blue flowers. The toxic part is the leaf. **Nicotine** itself is a colourless, volatile, bitter tasting liquid.

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Uses

1. Tobacco which can be smoked, sniffed, or chewed, is a respiratory and CNS stimulant.
2. Nicotine is a powerful pesticide.

Clinical Features

Acute Poisoning [18]

- Salivation, nausea, vomiting, diarrhoea. Characteristic odour of tobacco may be perceived in the breath.
- Vertigo, headache, sweating, confusion, ataxia, convulsions.
- Hypotension, tachycardia (followed by bradycardia), cardiovascular collapse.

Chronic Poisoning

- Addiction to tobacco results in tremor, insomnia, anxiety, palpitations, headache, amblyopia, and an increased tendency to bronchitis, oral and lung cancer, heart disease, Buerger's disease, etc.

Treatment

Acute Poisoning

- Decontamination (in case of ingestion).
- Cardiac monitoring.
- Atropine for bradycardia and hypotension.
- Diazepam for convulsions.
- Supportive measures.

Chronic Poisoning

- Withdrawal of tobacco use.
- Psychological counselling.

Usual Fatal Dose

Tobacco: 20 gm.
Nicotine: 60 mg.

Autopsy Features

1. Brownish froth at mouth and nostrils.
2. Congestion of g.i.tract, with brownish discolouration of gastric mcosa (in ingestion).
3. Odour of tobacco.
4. Dark coloured blood.
5. Pulmonary oedema.

Forensic Significance

- Most cases of poisoning are accidental, arising out of the use of nicotine as a pesticide, as also from the abuse of tobacco.
- Tobacco is one of the commonest drugs of abuse worldwide.

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CHAPTER - 12

Asphyxiants

The general expression for irrespirable or toxic gases is **asphyxiants**, which may be classified as follows:

1. **Simple Asphyxiants** – These gases displace oxygen from the ambient air and reduce the partial pressure of available oxygen. Examples include carbon dioxide, nitrogen, aliphatic hydrocarbon gases (butane, ethane, methane, and propane), and noble gases (argon, helium, neon, radon, and xenon).

2. **Respiratory Irritants** – These gases damage the respiratory tract by destroying the integrity of the mucosal barrier. Examples include ammonia, chlorine, formaldehyde, hydrogen sulfide, methyl isocyanate, oxides of nitrogen, ozone, phosgene, and sulfur dioxide.

3. **Systemic Asphyxiants** – These gases produce significant systemic toxicity by specialised mechanisms. Examples include carbon monoxide, cyanide, and smoke.

Only ammonia, carbon monoxide, cyanide, and methyl isocyanate will be discussed.

AMMONIA

Physical Appearance

Extremely irritant gas with a penetrating odour. It is highly water soluble (forming ammonium hydroxide which is an alkaline corrosive).

Uses

- Agriculture (fertilizer).
- Mining.
- Manufacture of plastics and explosives.
- Refrigerant.
- Cleaning and bleaching agent.
- Treatment of syncope in the form of smelling salts.

Usual Fatal Dose

5 to 10 ml of liquid ammonia. Inhalation of the gas at concentrations above 5000 ppm can be rapidly fatal.

Clinical Features

- Inhalation produces such severe upper airway irritation that the victim seldom remains exposed for more than an instant, unless he is trapped. Symptoms include lacrimation, cough, dyspnoea, convulsions, coma, and death. There is glottic and laryngeal oedema, sloughing of bronchial mucosa, and chemical pneumonitis with pulmonary oedema.

- Ingestion of ammonia solution produces corrosion of the alimentary tract and aspiration pneumonia.

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- Dermal contact can result in deep, penetrating burns.
- Ocular exposure can result in immediate and serious chemical burn with rapid penetration into the interior of the eye. Permanent impairment of vision or even blindness may result.

Diagnosis

- Chest X-ray in dyspnoeic patients.
- Early endoscopy to determine the extent of injury.
- Barium swallow after 1 to 2 weeks to rule out oesophageal strictures.
- Presence of ammonia in an unknown solution, stomach contents, or vomitus can be confirmed by placing an open bottle of conc. HCl in the vicinity. This will produce copious white fumes of ammonium chloride.

Treatment

- Ocular exposure should be treated with prolonged irrigation with water.
- Dermal exposure requires washing with soap and water, followed by copious irrigation with water alone.
- Inhalation should be treated with oxygen, PEEP (positive end expiratory pressure), intubation, and bronchodilators.
- In the case of ingestion, a small quantity of water or milk can be administered as a first-aid measure to dilute the chemical. Demulcents can be given. Stomach wash and emetics are contraindicated.

Forensic Significance

While poisoning with ammonia is not very common, most of the cases reported are suicidal in nature. Since the solution or gas even when weak has a distinct irritant smell, accidental poisoning is unlikely. Obviously, its properties preclude its choice for murder. However, of late ammonia is being used as a spray to incapacitate victims of robbery. Serious eye injuries can result.

METHYL ISOCYANATE (MIC)

MIC is one of a group of isocyanates, the others being toluene diisocyanate (TDI) and diphenylmethane diisocyanate (MDI).

Physical Appearance

Colourless liquid with a sharp odour, which becomes gaseous at 39°C. It is an extremely reactive chemical and needs to be stored carefully. Contact with water results in an exothermic reaction.

Uses

1. Manufacture of carbaryl (a carbamate pesticide).
2. Manufacture of polyurethane articles (plastics, urethane foam, adhesives, etc).

Mode of Action

MIC is a powerful respiratory irritant.

Clinical Features

Inhalation of MIC gas produces immediate lacrimation, photophobia, and corneal ulceration ; later, cough, dyspnoea, chest pain, haemoptysis, and pulmonary oedema ; less commonly, vomiting, convulsions, and coma. Dermal exposure results in erythema and vesiculation.

Long term effects after survival include RADS (reactive airways dysfunction syndrome), and pregnancy-related problems: high incidence of spontaneous abortions, and increased perinatal mortality.[1,2]

Treatment

1. Decontamination of skin and eyes with saline.
2. Oxygen.
3. Bronchodilators and corticosteroids may be beneficial.
4. Supportive measures.

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Isocyanates are not the same as cyanides, and antidotes for the latter such as nitrites and sodium thiosulfate must not be used for the former.

Autopsy Features

1. Signs of asphyxia.
2. Pulmonary and cerebral oedema.
3. Generalised visceral congestion.

Forensic Significance

MIC was involved in one of the most devastating gas disasters, which occurred in Bhopal, Madhya Pradesh in 1984, leaving more than 2000 people dead (unofficial estimates put the figure at more than 10,000), and more than 200,000 injured.[3] The incident occurred in a small pesticides division of Union Carbide Company manufacturing carbaryl (a carbamate), for which methyl isocyanate is required. This deadly chemical was stored in huge, double-walled stainless steel tanks, one of which burst on the night of December 2, 1984. More than 24,000 Kg of MIC gas escaped over the next several hours into the atmosphere forming an ominous white cloud that drifted rapidly over the surrounding heavily populated neighbourhood killing thousands in their sleep and incapacitating several thousands more.

CARBON MONOXIDE (CO)

Physical Appearance

Pure carbon monoxide is an odourless, colourless, non-irritating gas, which is lighter than air.

Sources

- Incomplete combustion of almost any form of fuel (wood, charcoal, gas, kerosene).
- Automobile exhaust.
- Fires.
- Tobacco smoke.

Endogenous CO resulting from haeme degradation can never reach toxic levels on its own. Normal CO level in plasma is in the range of 1 to 5 % and may rise upto 7 to 8 % in smokers.

Usual Fatal Dose

This is usually expressed in terms of plasma concentration of the gas (carboxyhaemoglobin or COHb). COHb level exceeding 50 to 60 % is potentially lethal.

Toxicokinetics

The lungs avidly absorb CO which combines with haemoglobin (85%) and myoglobin (15%). Elimination occurs exclusively through the lungs.

Mode of Action

Carbon monoxide has an affinity for haemoglobin which is 230 to 270 times greater than that of oxygen. Therefore, in spite of adequate partial pressure of oxygen (PO_2) in blood, there is reduced arterial oxygen content. Further, CO causes a leftward shift of the oxyhaemoglobin dissociation curve, thus affecting the offloading of oxygen from haemoglobin to the tissues. The net result of all this is the decreased ability of oxygen to be carried by the blood and released to tissues.

Clinical Features

Acute Poisoning:

Mentioned in relation to severity of exposure in **Table 12.1**. The earliest manifestations are often non-specific and may be confused with other conditions. **Table 12.2** outlines the important conditions in the differential diagnosis.

Two of the "classical" features of CO poisoning mentioned in several textbooks on toxicolgy are actually quite rarely encountered in clinical practice:

- Cherry red colour of blood and tissues (including skin) is seen only in 2 to 3 % of cases.[4]
- Development of cutaneous bullae (blisters) is another uncommon finding in clinical practice.[5]

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Chronic Poisoning:

The following features are seen in chronically poisoned patients:

1. Headache, dizziness, confusion.
2. Weakness, nausea, vomiting, abdominal pain.
3. Paraesthesias.
4. Visual disturbances.
5. Hypertension, hyperthermia.
6. Cherry red skin.
7. Palpitations, aggravation of angina.

Table 12.1 Acute carbon monoxide poisoning

Severity	Symptoms and Signs
Mild (COHb < 30%)	Headache, nausea, vomiting, dizziness, exertional dyspnoea
Moderate (COHb 30 to 40%)	Chest pain, blurred vision, confusion, weakness, increasing dyspnoea, tachycardia, tachypnoea, ataxia
Severe (COHb > 40%)	Trismus, muscle spasms, convulsions, palpitations, disorientation, ventricular dysrhythmias, hypotension, myocardial ischaemia, respiratory failure, coma

Table 12.2 Differential diagnosis of carbon monoxide poisoning

Alcoholic intoxication	Hyperventilation syndrome
Cerebrovascular accident	Meningitis, encephalitis
Migraine	Epilepsy
Food poisoning	

Diagnosis

1. *Estimation of carboxyhaemoglobin level* (COHb) : Normal levels range from 0 to 5%, but in heavy smokers it may be as high as 10%.[6]

2. *Pulse oximetry:* It is a non-invasive method of measuring oxygen saturation and is relatively easy to perform, painless, rapid, and accurate. A special sensor is placed on a patient's finger, toe, or nose. The sensor consists of a light-emitting diode that projects two discrete wavelengths of light corresponding to saturated and unsaturated haemoglobin (660 and 940 nm) together with a photodetector.

3. *Arterial blood gases:* Partial pressure of oxygen is usually normal, but the oxygen saturation expressed as a percentage is decreased. Metabolic acidosis is invariably present.

4. *ECG:* This may reveal myocardial damage in the form of ST depression or elevation, T-wave flattening or inversion, and dysrhythmias.

5. *Chest X-ray:* This may reveal ground-glass appearance, perihilar haze, peribronchial cuffing, and intra-alveolar oedema.

6. *Bedside Tests:*
   a) Take 1 drop of blood and dilute with 10 to 15 ml of water. Compare with normal blood diluted in the same manner. Blood containing carbon monoxide is pink.
   b) Dilute 1 ml of the patient's blood with 10 ml of water in a test tube and add to it 1 ml of a 5% solution of sodium hydroxide. If COHb is present, the solution will turn straw yellow (< 20% COHb) or pink (> 20% COHb). In the case of normal blood (HbO_2) the solution turns brown in colour.

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Treatment

1. Immediate removal from the contaminated environment.
2. Oxygen (100%) through a tight-fitting mask or endotracheal tube, until COHb falls to 15 to 20%.
3. Monitor cardiac and respiratory status.
4. Watch for the development of cerebral oedema with serial neurologic exams, CAT scans, and fundoscopic examination. Hyperventilation (PCO_2 25 to 30 mm Hg), head elevation (35^0), and mannitol (0.25 to 1 gm/Kg of 20% solution over 30 minutes) are recommended as initial management of raised intracranial pressure. The role of corticosteroids is controversial.
5. Metabolic acidosis must not be treated aggressively. Sodium bicarbonate is not recommended.
6. Convulsions can be controlled with i.v diazepam or phenytoin in the usual manner.
7. Physical activity should be restricted for at least 1 month after the exposure to minimise the incidence of cerebral demyelination.
8. **Antidote:** Several authorities consider administration of hyperbaric oxygen (HBO) to be antidotal in its effects in carbon monoxide poisoning. It involves inhalation of oxygen at a pressure greater than 1 atmosphere absolute (ATA). Studies among human victims of CO poisoning indicate significantly reduced incidence of neuropsychiatric symptoms in those treated with HBO as compared with those who receive normobaric oxygen.[7]

It must be borne in mind however, that HBO therapy is asociated with serious risks such as cerebral gas embolism, rupture of tympanic membranes, visual deficits, and oxygen toxicity (convulsions and pulmonary oedema). So the routine administration of HBO is not recommended in every case of CO poisoning.

Autopsy Features

- Cherry red (pink) colour of skin, especially noticeable in the areas of postmortem lividity.
- Cutaneous bullae (skin blisters) are sometimes seen in the regions of the calves, buttocks, wrists, and knees.
- Cherry pink colour of blood and tissues. If blood is diluted with water in a test tube and held against light or a white background, the pink colour will be more easily made out.
- Pulmonary oedema.
- In delayed deaths, necrosis and cavitation of basal ganglia, especially globus pallidus and putamen are commonly described features.

It is mandatory to collect blood for chemical analysis preferably from a peripheral vein. It is advisable to seal off the sample of blood with a layer of liquid paraffin on top. Sodium fluoride may be added as a preservative.

Forensic Significance

Next to carbon dioxide, carbon monoxide is the most abundant atmospheric pollutant and is progressively increasing in concentration.[8] Apart from its role as an environmental contaminant,CO is responsible for a significant number of deaths encountered in forensic practice.[9] Once upon a time when domestic gas consisted of coal gas (which contained upto 7% CO), suicides accomplished with it at home were very common in Western countries. "Putting the head in the gas oven" was the most common form of self-destruction in countries such as the UK. Now that coal gas has been replaced by natural gas (which contains little or no CO), a major means of domestic suicide has been removed. But incomplete combustion of natural gas can release CO which can cause accidental poisoning in ill-ventilated areas.

Today the suicidal use of CO is utilised in a different way. The victim utilises the exhaust fumes

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of a motor car either by merely sitting in a closed garage with a window of the car open while the fumes build up in the enclosed area, or a device is fitted (e.g., a hose) to pipe the gas into the interior of the car with all windows rolled up.

Accidental CO poisoning can occur in several other situations apart from domestic exposure. Defective exhaust system of an automobile can allow gas to percolate through the floor or engine bulkhead into the interior. Sometimes the driver may become so affected that he loses control of the vehicle resulting in a crash.

A common cause of accidental CO poisoning resulting in mass deaths is a conflagration where-in a large building (hotel, theatre, block of flats, etc) goes up in flames. The majority of deaths in such cases are caused by inhalation of smoke (containing CO) rather than by burns.

Homicidal poisoning with CO is rare, but cases have been reported (and continue to be reported) from time to time. [10]

CYANIDE

Physical Appearance

Cyanide occurs as a gas or liquid or solid. In its gaseous state it is referred to as hydrogen cyanide (HCN); the liquid form is referred to as hydrocyanic acid or Prussic acid ; salts of cyanide occur as solids (white, crystalline powder).

The odour of cyanide, especially the gas, is described as "bitter almond" in nature. However, it cannot be perceived by everybody. 20 to 40 % of the human population (mostly males) do not possess this capacity which is inherited as a sex-linked recessive trait. Some sources put this at 40 to 60 %.[11]

Uses

1. *Industrial:* electroplating, metal processing, photographic processes, production of synthetic rubber, and manufacture of plastics.
2. *Agriculture:* insecticide and rodenticide.

Table 12.3 Cyanogenic plants

Plant	Toxic Part	Cyanogenic Glycoside
Prunus species: cherry laurel, chokeberry, mountain mahogany, bitter almond, peach, apricot, plum, and wild black cherry	Leaf, bark, seed	Prunasin or amygdalin
Sorghum species: sorghum, sudan grass, johnson grass, and arrow grass	Grain, shoot	Dhurrin
Apple, pear, crab apple	Seed	Amygdalin
Cassava, lima beans	Bean, root	Linamarin
Miscellaneous: christmas berry, velvet grass jet berry bush, elderberry, bamboo, cycad nut	Bead, leaf, shoot, sprout	Unclear

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3. *Medicinal:*
   a) Laetrile (synthetic amygdalin) is used as a chemotherapeutic agent for cancer in the USA though studies have shown it is not efficacious, and in fact can be hazardous.[12]
   b) Sodium nitroprusside is an effective antihypertensive and is especially useful in treating hypertensive crisis as an intravenous infusion. But it is metabolised in the body to cyanide and infusions exceeding the recommended dose can lead to cyanide toxicity.[13]
4. *Laboratory:* cyanide is used in various laboratory processes.

Sources

1. *Plants:* cyanide is present in the form of cyanogenic glycosides in a wide variety of plants and plant parts (Table 12.3). Hydrolysis of these glycosides by digestive enzymes can release cyanide in the g.i tract.
2. *Combustion:*
   a) Burning of plastic furniture (polyurethane or polyacrylonitrile).
   b) Burning of silk or wool.
   c) Cigarette smoking

Usual Fatal Dose

Hydrogen cyanide: Inhalation of 1 part in 2000 can kill instantaneously, 1 part in 10,000 within a few minutes, 1 part in 50,000 within a few hours. The upper limit of safety is 1 part in 100,000.

Hydrocyanic acid : 50 to 100 mg.
Cyanide salts (of sodium, potassium, or calcium): 100 to 200 mg.

Toxicokinetics

Absorption is rapid across both skin and mucous membrane. Ingestion of cyanide salts results in the release of HCN through the action of hydrochloric acid in the stomach, and is subsequently absorbed as the cyanide ion (CN^-). Metabolism occurs mainly in the form of conversion to thiocyanate by the enzyme rhodanese (present in the mitochondria of liver and kidneys), which needs sodium thiosulfate for effective functioning. Some of the cyanide is converted to cyanacobalamin (vitamin B_{12}) in the presence of hydroxocobalamin (vitamin B_{12a}). Small amounts of cyanide are excreted in the breath and sweat producing the characteristic bitter almond odour.

Mode of Action

The toxic effect of cyanide is mainly attributed to its production of a histotoxic anoxia by inhibition of cytochrome oxidase.[14] This is a metalloenzyme essential for oxidative phosphorylation which is responsible for aerobic energy production. Cytochrome oxidase functions in the electron transport chain within mitochondria converting catabolic products of glucose into adenosine triphosphate (ATP). Cyanide inhibits cytochrome oxidase at the cytochrome aa3 portion of the enzyme. As a result of the consequent reduced ATP production, tissues resort to anaerobic energy production which is a less efficient alternative pathway for formation of ATP. Pyruvic acid no longer enters the krebs cycle, but is converted to lactic acid which accumulates and results in metabolic acidosis.[15]

Clinical Features

Acute Poisoning: Inhalation produces the most rapid and serious exposures resulting in almost immediate coma, while ingestion causes less rapid onset because of slower entry into the circulation, and passage of cyanide through the portal system where the liver metabolises some of it by the first-pass effect.

1. CNS : headache, anxiety, agitation, confusion, convulsions, and coma. Pupils are often dilated.
2. CVS : initial bradycardia and hypertension, followed by tachycardia and hypotension and ventricular dysrhythmias.

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3. RS: tachypnoea followed by bradypnoea, and cardiogenic or non-cardiogenic pulmonary oedema.
4. GIT: ingestion of cyanide salt produces nausea, vomiting, and abdominal pain.
5. Skin: brick-red colour of skin and mucous membranes is said to be characteristic.[16] It is due to increased haemoglobin oxygen saturation in venous blood because of decreased utilisation of oxygen by tissues.

   The skin feels cold and clammy to the touch. Cyanosis is a late feature.

Chronic Poisoning: Chronic, low-level exposure may result in any of the following

1. *Tobacco amblyopia:* progressive loss of visual function seen almost exclusively in heavy smokers.
2. *Leber's hereditary optic atrophy:* congenital deficiency of rhodanese is suspected in this condition which exclusively affects males and results in acute visual failure due to the sensitivity of optic nerve to cyanide.
3. *Tropical ataxic neuropathy:* it is prevalent among populations consuming large quantities of cassava or tapioca (manihot). This tuber contains two cyanogens – linamarin and lotaustralin which can be removed only by proper fermentation techniques. Symptoms include peripheral sensory neuropathy, optic atrophy, ataxia, deafness, glossitis, stomatitis, and scrotal dermatitis.

Diagnosis

1. Characteristic odour in the vicinity of the patient.
2. **Lee-Jones test:**
   - Add a few crystals of ferrous sulfate to 5 ml of gastric aspirate.
   - Add 5 drops of 2% sodium hydroxide.
   - Boil and cool.
   - Add 10 drops of 10% hydrochloric acid.
   - Interpret.

   Greenish-blue colour indicates cyanide, while purplish colour indicates salicylates.

3. Serum cyanide level : this is confirmatory, but difficult to accomplish in practice. Normal serum level is less than 0.004 mg/ml for non-smokers, and 0.006 mg/ml for smokers.

Treatment

Time is of the essence ! Do not delay !

1. **Stabilisation:** assisted ventilation, 100% oxygen, cardiac monitoring, i.v access, treatment of metabolic acidosis, vasopressors for hypotension.

2. **Decontamination:**
   a) Cutaneous exposure - remove clothing and wash skin with soap and water.
   b) Ingestion - stomach wash (preferably with 5% sodium thiosulfate solution), activated charcoal, and cathartics, after antidotal therapy has been instituted.

3. **Antidotal therapy:**
   a) The 3-step *Eli Lilly cyanide kit* approach[17]
   First step: Amyl nitrite (one perle of 0.2 ml is crushed and inhaled for 30 seconds) every minute until the 2nd step is begun.

   Second step: Sodium nitrite (3% solution) slow i.v., i.e., over 5 to 10 minutes.

   Adult dose – 10 ml (300 mg).
   Paediatric dose – 0.33 ml/kg, upto a maximum of 10 ml.

   Exceeding the recommended dose can result in fatal methaemoglobinaemia.

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Third step: Sodium thiosulfate (25% solution), 3 to 5 ml/min, i.v.

Adult dose - 50 ml (12.5 gm).
Paediatric dose - 1.65 ml/kg (412.5 mg/kg), upto a maximum of 50 ml.

Mechanism of action of nitrites : Nitrites induce methaemoglobinaemia which causes the detachment of cyanide from the haeme group of cytochrome oxidase.

Mechanism of action of sodium thiosulfate: It enables the enzyme rhodanese to catabolise cyanide to non-toxic thiocyanate which is excreted in the urine.

b) *Other Antidotes* -

i) **4-dimethylaminophenol** (4-DMAP) : It is the agent of choice to induce methaemoglobinaemia in Europe (as opposed to the USA where nitrites are more popular). Dose : 3 mg/kg, i.v.

ii) **Dicobalt edetate** (Cobalt-EDTA) : It acts by chelating cyanide without inducing methaemoglobinaemia. Cobalt-EDTA is used in Britain and France under the brand name Kelocyanor. It is unfortunately associated with serious adverse effects including hypotension, cardiac arrhythmias, decreased cerebral blood flow, and angioedema.[18] In fact the edetate (ethylene diamine tetra acetate) part of the antidote is included only because it is hoped that it will minimise the toxicity of cobalt. Dose : 20 ml, i.v., (300 to 600 mg).

iii) **Hydroxocobalamin** (Vitamin B_{12} precursor): It combines with cyanide to form cyanacobalamin (vitamin B_{12}), which is excreted in the urine. Dose: 50 mg/kg of commercial solution (1000 mg/ml). This may require the i.v infusion of upto 3.5 litres in an adult!

iv) **Alpha-ketoglutaric acid** : It is presently only in the experimental stage, but shows a great deal of promise since it binds with cyanide to render it non-toxic without inducing methaemoglobinaemia.[19]

v) Pyruvate, mercaptopyruvate, sulfur sulfanes, and stroma-free methaemoglobin solutions have been tried in animal studies, but are not yet recommended for human use.

Methylene blue is NOT an antidote for cyanide and must NOT be used.

Autopsy Features

External:

1. Odour of bitter almonds.
2. Brick red colour of skin and mucous membranes.
3. Cyanosis of extremities.
4. Froth at mouth and nostrils (may be blood-stained).

Internal:

1. Haemorrhagic gastritis (ingestion death).
2. Pulmonary and cerebral oedema.
3. Disseminated petechiae in brain, meninges, pleura, lungs, and pericardium.

The most appropriate fluids and tissues to remove for chemical analysis are blood, stomach contents, lung, liver, kidney, brain, heart, and spleen.[20] Lung should be sent intact, sealed in a nylon bag. Spleen is said to be the best specimen for cyanide analysis since it generally has the highest concentration of the poison owing to a strong presence of RBC.

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Forensic Significance

Homicide: The very mention of cyanide to a lay person would make him think of murder. Like arsenic and strychnine, cyanide has a reputation (quite unfounded) of being a homicidal poisoner's favourite, probably because of the perpetuation of such a notion in popular detective fiction. But the reality is that except for certain exceptional situations, its employment in murder has been quite rare.

There have been a few cases over the years, but two features go against the concept of cyanide being an ideal homicidal poison – its possible detection by smell, and the suspicion likely to be aroused by the dramatic nature of death. Cyanide in fact has been more commonly involved in the commission of mass murder, for e.g., the genocide of Jews perpetrated by the Nazis during the second world war. Initially the Nazis used carbon monoxide, but later in order to expedite their gory task they began employing hydrogen cyanide (zyklon B). Upto 10,000 innocent people per day were butchered by this "efficient" gas and the final tally ran into millions.

More recently, mass homicide (albeit on a much smaller scale) was accomplished with the help of cyanide by Jim Jones, a self-styled preacher who founded a cult called the People's Temple in 1974, in California, USA. This "religious" sect comprising mainly mentally afflicted individuals, cripples, drug addicts, and ex-convicts, soon moved to Guyana due to local public disapproval. In November 1978, most of them (numbering around 900) died after drinking a cyanide solution prepared by Dr.L.Schat, a medical officer of the cult on instructions issued by Jones. The latter shot himself to death. The reason for such an abrupt and bizarre end to this cult is unclear, though it may have been triggered off by rumours of imminent investigations into the sect's activities by a group of relatives of some cult members.

A different type of mass poisoning resulted in 1982 in Chicago, USA, when paracetamol capsules (Tylenol) were deliberately tampered and laced with hydrogen cyanide. Seven innocent victims died. In 1986, capsules of another brand of analgesic (Excedrin) were tampered and laced with cyanide by a woman, Stella Nichols whose intended victim was her own husband, Bruce Nichols. She succeeded, but also inadvertently killed another victim in the process (Susan Snow). Stella was convicted and sentenced to life imprisonment on May 9, 1988.

Cyanide has been (and continues to be) used legitimately to kill convicted criminals in some of the states of the USA, gassing with it being the official mode of execution in these states.

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CHAPTER - 13

Antipyretics

"A rule of thumb in the matter of medical advice is to take everything any doctor says with a grain of aspirin!" – **Goodman Ace.**

While there are numerous antipyretic-analgesics available in the market today, two of the commonest drugs which have been around a long time, and which continue to be used extensively world-wide are **acetylsalicylic acid** and **paracetamol.**

ACETYLSALICYLIC ACID

Common Name
Aspirin

Characteristics
Acetylsalicylic acid belongs to a group of compounds referred to as **salicylates.** These compounds are derivatives of salicylic acid and include apart from acetyl salicylic acid, also sodium salicylate and methyl salicylate. Salicin, a naturally occurring salicylate is a constituent of several plants but is present in highest concentration in the willow tree (*Salix alba vulgaris*), which grows near lakes and rivers in temperate climates and whose branches are used to make cricket bats and baskets.[1]

Acetyl salicylic acid is an odourless, white, crystalline powder with an unpleasant saline taste.

Uses
1. Antipyretic.
2. Analgesic.
3. Treatment of rheumatoid arthritis.
4. Low-dose aspirin is used in the prophylaxis of cerebrovascular ischaemic events,[2] angina pectoris,[3] and is also recommended by some authorities for the prevention of colon cancer,[4] and migraine.[5]

Usual Fatal Dose
Salicylic acid: 70 to 80 gm.
Sodium salicylate, aspirin: 15 to 20 gm.
Methyl salicylate: 5 to 15 ml.

Toxicokinetics
Salicylates are rapidly absorbed from the stomach, and to a slightly lesser extent from the small intestine. Delayed absorption is seen in the following situations : enteric coated preparations, pylorospasm, pyloric stenosis, and bezoar formation. Therapeutic serum salicylate levels should not exceed 30 mg/100 ml. Metabolism occurs chiefly in the liver, where salicylates are broken down into salicyluric acid, ether glucoronide, ester glucoronide, and gentisic acid. Excretion is mainly through urine.

The half-life of salicylates is 2 to 4 hours at therapeutic levels, but may increase to 20 hours at toxic

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levels. There is also a decrease in protein binding from 90% at therapeutic levels to less than 75% at toxic levels.[6] The apparent volume of distribution increases from 0.2 litres/kg to more than 0.3 L/kg.

Mode of Action

Salicylates stimulate the respiratory centre in the brain stem leading to hyperventilation and respiratory alkalosis. They also interfere with Krebs cycle, inhibit production of ATP, and increase lactate production, leading to ketosis and a wide anion-gap metabolic acidosis. In children respiratory alkalosis is quite transient, and metabolic acidosis is the predominant feature.[7]

Salicylates are extremely irritating to the g.i mucosa, and overdose often results in haemorrhagic gastritis.

Clinical Features

Acute Poisoning

A. *Early* – nausea, vomiting, sweating, tinnitus (ringing or hissing), vertigo, and hyperventilation due to respiratory alkalosis.

B. *Late* – deafness, hyperactivity, agitation, delirium, convulsions, hallucinations, hyperpyrexia. Coma is unusual.

C. *Complications* – metabolic acidosis, pulmonary oedema, rhabdomyolysis, cardiac depression, thrombocytopenic purpura. Salicylates must not be therapeutically administered to children under 15 years of age, especially if they are suffering from chicken pox or influenza. There is a serious risk of precipitating *Reye's syndrome* which can be fatal. **RDK Reye**, an Australian paediatrician first described this condition in 1963.[8] Reye himself could not elucidate the aetiology, but subsequent investigators associated the syndrome with viral illnesses and apirin. The main features are acute onset of hepatic failure and encephalopathy. It probably results from damage to mitochondria in liver cells.[9] Recovery is associated usually with permanent neurological sequelae.

Chronic Poisoning: (Salicylism)

This is characterised by slow onset of confusion, agitation, lethargy, disorientation, slurred speech, hallucinations, convulsions, and coma. There may also be tinnitus, hearing loss, nausea, dyspnoea, tachycardia, and fever.

Diagnosis

Laboratory Findings:

1. Anion-gap acidosis.
2. Hypokalaemia (acidosis may mask it).
3. Hypocalcaemia.
4. Hypoglycaemia.

Bed-side Tests:

1. *Ferric chloride test:* Add a few drops of 10% ferric chloride solution to 1 ml of urine. A purple colour indicates the presence of salicylates. However it is not conclusive, since a positive result is also obtained in phenol, phenothiazines, phenylbutazone, and oxyphenbutazone.

2. *Trinder's test:* Reagent : Trinder's reagent is used which is obtained by mixing 40 gm of mercuric chloride (dissolved in 850 ml of purified water), with 120 ml of aqueous HCl (1 mol/L) and 40 gm of hydrated ferric nitrate, followed by dilution to 1 litre with purified water.

   Method : The test can be done on urine, stomach contents, or scene residue. Add 0.1 ml of Trinder's reagent to 2 ml of sample and mix for 5 seconds. A strong violet colour indicates the presence of salicylates. Mere darkening is not significant.[10] If the sample to be tested is stomach contents or scene residue, it is better to first boil 1 ml of the sample with 1 ml of

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aqueous HCl (0.1 mol/L) for 10 minutes, cool, filter, and then neutralise with 1 ml of aqueous sodium hydoxide (0.1 mol/L).[11]

Confirmatory Test

The only confirmatory test is to estimate the serum salicylate level. Unfortunately, the seriousness of poisoning correlates poorly with serum levels. Previously, the Done nomogram (first published in 1960) was highly recommended to correlate serum salicylate level with the degree of intoxication at varying intervals after acute ingestion of aspirin.[12] But there are severe limitations to its use and is now not generally considered to be reliable. Studies have indicated that it has poor predictive value.[13]

Treatment

1. *Stomach wash* may be beneficial upto 12 hours after ingestion, since toxic doses of salicylates often cause pylorospasm and delayed gastric emptying.[14]

2. *Activated charcoal (AC)*: It is said to be very efficacious in the treatment of salicylate poisoning since each gram of AC can adsorb 550 mg of the drug. A 10 : 1 ratio of AC to salicylate ingested appears to result in maximum efficiency.

3 *Urinary alkalinisation:* This should not be confused with forced diuresis which was recommended in the past, where the accent was on increasing urinary flow rate in order to increase salicylate clearance. It caries with it the risk of fluid overload with attendant complications.

   Alkalinisation of both blood and urine can be achieved with intravenous sodium bicarbonate.*

   Acetazolamide must not be used since it produces a systemic metabolic acidosis.

4. *Haemodialysis:* It is very effective in salicylate poisoning and must always be considered in the presence of cardiac or renal failure, intractable acidosis, convulsions, severe fluid imbalance, or a serum salicylate level more than 100 mg/100 ml.

5. *Supportive measures*:
   a) Correction of fluid and electrolyte imbalance (watch out for fluid overload!).
   b) Hypoprothrombinaemia can be corrected by 2.5 to 5 mg of vitamin K i.v every day.
   c) Hyperpyrexia must be tackled by cooling measures (for e.g., ice in the axilla and groin).
   d) Correction of metabolic acidosis with $NaHCO_3$.
   e) Correction of hypocalcaemia with calcium gluconate i.v (5 to 10 ml in adults).
   f) Correction of hypoglycaemia with glucose i.v (50 ml of 50% dextrose or 1 ml/kg).
   g) Treatment of convulsions with benzodiazepines.

6. *Treatment of Reye's syndrome*[15]:
   a) Admit the patient to an intensive care unit.
   b) Raise the head-end of bed (40^0).
   c) Mannitol i.v (0.2 to 1.0 gm/kg.).
   d) Acute hyperventilation may be helpful.
   e) Short acting barbiturates in resistant cases.

Autopsy Features

- Petechiae in the skin (occasionally).
- Erosions of gastric mucosa. Black, altered blood may lie in the stomach. Sometimes massed concretions of tablets are present.
- Petechiae in various organs and serous membranes (parietal pleura, pericardium).
- Pulmonary and cerebral oedema.

*Do not give sodium bicarbonate orally since it can enhance salicylate absorption by facilitating dissolution.

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Forensic Significance

Most of the cases of overdose are suicidal in nature; a few may be accidental. In some individuals, a small dose of aspirin can provoke a fatal hypersensitivity reaction. The patient is usually (curiously) a middle-aged female, and often has nasal polyps. Within minutes of ingestion there is an acute vasomotor rhinitis, angioneurotic oedema, and urticaria. Death results from laryngeal oedema, hypotension, or shock.

PARACETAMOL

Synonym

Acetaminophen

Uses

1. Antipyretic : In 1893, **von Mering** introduced paracetamol, and a hundred years later it is still going strong. In fact, paracetamol is the active metabolite of both acetanilide and phenacetin.
2. Analgesic

Usual Fatal Dose

20 to 25 gm. However doses as low as 10 gm can cause serious hepatotoxicity. Children under the age of 10 years appear to be more resistant to the toxic effects of paracetamol.[16]

Toxicokinetics and Mode of Action

Paracetamol is rapidly and completely absorbed from the g.i tract. Peak plasma levels are reached in ½ to 1 hour, while the plasma half-life is about 2 hours. Normally about 90% of the drug undergoes hepatic conjugation with glucuronide and sulfuric acid to form inactive and harmless metabolites, while 10% is oxidised (through P450 mediation) to **N-acetyl-p-benzoquinoneimine (NAPQI)** which is a highly reactive intermediate. NAPQI is capable of covalent binding and arylating critical cell proteins inducing a series of events that result in cell death. In the normal course, glutathione rapidly detoxifies this intermediate to cysteine and mercapturate conjugates. In the overdose situation, glutathione stores become depleted and the toxic NAPQI binds covalently with hepatocytes of the liver causing centrilobular hepatic necrosis.

Clinical Features

Acute Poisoning:

- *Stage I* (1/2 hr to 24 hrs) : Anorexia, vomiting, sweating, malaise.
- *Stage II* (24 to 72 hrs) : Relatively symptom-free. There may be right upper quadrant pain. Liver function tests may be abnormal.
- *Stage III* (72 to 96 hrs) : Hepatic necrosis sets in with coagulation defects, jaundice, and encephalopathy. Nausea and vomiting reappear. Renal failure and myocardial damage are frequently present. Death is usually due to hepatic failure and is preceded by coma.
- Stage IV (4 days to 2 wks) : If the patient survives the IIIrd stage, complete resolution of hepatic damage is the rule rather than the exception. There are no reported cases of chronic hepatic dysfunction from paracetamol.

Chronic Poisoning:

This is uncommon, but cases have been reported where-in an individual has consumed large doses of paracetamol over a period of time for relief of chronic pain which resulted in toxic hepatitis.

Chronic overdose among children is more common than in adults mainly because of dose miscalculation by parents. Features include anorexia, vomiting, lethargy, low body temperature, hepatomegaly, and oliguria.

Diagnosis

1. *Evidence of hypoglycaemia, metabolic acidosis.*

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2. *Evidence of hepatocellular injury*: Elevated aspartate aminotransferase (AST), alanine aminotransferase (ALT), bilirubin, and prothrombin time.
3. *Evidence of renal damage*: Proteinuria, phosphaturia.
4. *Evidence of myocardial damage*: ECG changes indicative of dysrhythmias.
5. *Serum paracetamol level (PL)*: The lowest acute dose of paracetamol capable of toxicity is generally regarded as 7.5 gm in an adult and 150 mg/kg in a child, though it is more likely that the actual figures may be 15 gm and 250 mg/kg respectively.[17]

Interpretation of serum paracetamol levels is usually done on the basis of the **Rumack-Matthew nomogram.**

The purpose of the Rumack-Matthew nomogram is to predict the risk of hepatic injury at the outset and begin antidotal therapy well in time. Though the risk cannot be predicted earlier than 4 hours post-ingestion, this does not matter at all since there seems to be no added efficacy in antidotal therapy when begun earlier than 4 hours after the overdose.[18] In those cases where the time of ingestion is unknown and cannot be determined, it is advisable to determine both PL and AST. If the AST is elevated regardless of PL, begin antidotal therapy. If the PL is less than 10 mg/ml and AST level is normal, the antidote (N-acetylcysteine) can be withheld. If it is not possible for any reason to estimate the paracetamol level at all, then the risk of hepatic injury should be predicted on the basis of the history.

Treatment

1. **Stomach wash:** useful only in cases of very early presentation (< 1 hour), or in concomitant ingestion of other drugs which delay g.i absorption.
2. **Activated charcoal** can adsorb paracetamol, but it can also adsorb the antidote (N-acetylcysteine) and hence must be administered earlier to 4 hours post-ingestion.
3. **Anti-emetic,** if the patient is vomiting repeatedly.
4. **Supportive measures:**
   a) 10 to 20% dextrose for hypoglycaemia.
   b) Vitamin K if PT is elevated.
   c) Fresh-frozen plasma if there is overt bleeding.
   d) Mannitol (0.5 gm/kg given over 10 minutes) for cerebral oedema.
   e) Broad spectrum antibiotics i.v (ceftazidime or fluoxacillin) if necessary.
   f) H_2 antagonists to prevent upper g.i haemorrhage.
   g) Do not give sedatives, benzodiazepines, or NSAIDs.
5. **Antidotal therapy:**
   a) **Methionine:** This is an oral antidote that is popular in the UK and some other countries, but is presently not available in India. It is a glutathione precursor and protects against paracetamol-induced hepatic and renal damage, provided it is administered within 8 to 10 hours of ingestion. Dose : 2.5 gm, 4 doses, at 4-hour intervals.
   b) **N-acetylcysteine (NAC):** It is a derivative of L-cysteine, a naturally ocurring amino acid.

   Indications: NAC is indicated in paracetamol overdose if
   - PL estimated between 4 and 12 hours post-ingestion lies above the nomogram line.
   - Paracetamol ingested is more than 100 mg/kg.
   - Likelihood exists of paracetamol-induced hepatic failure.

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NAC is the antidote of choice for paracetamol poisoning in the USA, and is gaining in popularity elsewhere including India.[19] It gives maximum protection against hepatotoxicity when administered within 10 hours of paracetamol overdose, but can be given with (lesser) benefit upto 36 hours.

Dose: *Oral* – 5% solution given as loading dose of 140 mg/kg. This is followed by 17 more doses at 70 mg/kg, 4th hourly, total making upto 1330 mg/kg over 72 hours. The calculated dose can be administered mixed with water, or flavoured or carbonated drink.

Intravenous

i) 20 hour regimen → 150 mg/kg made up in 200 ml of 5% dextrose is given i.v over 15 minutes, followed by 50 mg/kg in 500 ml of 5% dextrose over 4 hours, and 100 mg/kg in 1 litre of 5% dextrose over 16 hours. The total dose works out to 300 mg/kg given over 20 hours.

ii) 48 hour regimen → useful in delayed admissions and massive ingestion. A loading dose of 140 mg i.v is given over 1 hour, followed 4 hours later by the first of 12 maintenance doses of 70 mg/kg, each administered over 1 hour.

Adverse effects: *Oral* - drinking NAC through a straw minimises its unpleasant odour. The main problem with oral NAC is induction of vomiting. Metoclopramide or ondansetron may have to be administered.

Intravenous – anaphylactoid reaction. If it occurs, it should be managed in the usual way with antihistamines, epinephrine, etc.

6. **Liver transplantation**: When fulminant liver failure develops after a massive paracetamol overdose, virtually the only treatment modality available is liver transplantation.

Autopsy Features

1. Liver may be enlarged and yellowish in colour. Microscopy reveals centrilobular necrosis.
2. Histological evidence of renal damage.
3. Cerebral oedema.

Forensic Significance

Attempted suicide with paracetamol accounts for a staggering 15 to 30% of cases of poisoning in the UK[20] ; a similar scenario exists in the USA where paracetamol overdose accounts for more hospitalisations than any other pharmaceutical agent.[21] While the situation is at present not as bad in India, there are alarming indications of rising incidence.[22]

Accidental poisoning most often results from inadvertent therapeutic overdose either as an acute or as a chronic phenomenon. A few cases result from hypersensitivity reactions which (though rare) may sometimes produce serious manifestations ranging from dermal to respiratory to anaphylactoid reactions.[23, 24]

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CHAPTER - 14

Food Poisons

To eat is human; to digest, divine.

- C.T. Copeland

Food borne toxins may cause isolated cases of poisoning, or a group of people may be involved simultaneously. In the latter, epidemiologic assessment is necessary. The following questions are particularly important:

1. Is the incubation period and nature of manifestations same in all the patients ?
2. What could be the mode of transmission ? (i.e., whether through food, water, contact, inhalation, etc.).
3. Are there any age or sex differences in the symptomatology ?
4. Which types of food or drink were served ? Can the offending item be identified ?
5. How was the food prepared /cooked ? How was it stored ?
6. What hygienic measures were taken or found lacking ?
7. Was there chemical or biological contamination ?

It may be necessary to inform the local Public Health Authority about the outbreak.

Common Sources of Food Poisoning

A. *Microorganisms*

I. **Bacterial Food Poisoning** : The common culprits include :

1. **Staphylococcus aureus**
   It is perhaps the commonest cause of food poisoning.
   Mode of action: Elaboration of an enterotoxin.
   Sources : Previously cooked proteinaceous food products: potato, meat, egg, milk, etc.
   Incubation period: 2 to 4 hours.
   Signs and symptoms: Vomiting, diarrhoea, abdominal pain.
   Diagnosis: 1. Clinical picture; 2. Stool culture
   Treatment: Supportive measures.

2. **Bacillus cereus**
   Mode of action : Elaboration of an enterotoxin.
   Sources: Fried rice, dried fruit, powdered milk, etc.
   Incubation period: 3 to 6 hours for emetic form, 8 to 16 hours for diarrhoeal form.
   Signs and symptoms: The illness may take one of two forms -
   1. Emetic form - characterised mostly by vomiting.
   2. Diarrhoeal form - mild looseness of stools.

   Diagnosis: Culture of contaminated food.
   Treatment: Supportive measures.

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3. **Shigella**

Mode of Action:

1. Invasion and destruction of small bowel mucosa.
2. Elaboration of enterotoxin and neurotoxin.

Sources: Fruits, vegetables, and milk. Shigella poisoning characteristically occurs in institutions.

Incubation period: 1 to 3 days.

Signs and symptoms: Usually affects young children. Main features include bloody diarrhoea, fever, bronchitis, and sometimes convulsions.

Diagnosis:

1. Stool culture.
2. Faecal leukocytes

Treatment:

1. Trimethoprim - sulfamethoxazole, or tetracycline, or ampicillin.
2. Supportive measures.

4. **Salmonella**

Mode of action: Invasion and destruction of small bowel mucosa.

Sources: Eggs, meat, and milk.

Incubation period & Signs and symptoms: There are four forms of the illness -

i) Gastroenteritis:
Incubation period: 12 hours to 2 days.
Main feature is watery diarrhoea stained with blood or mucus.
Diagnosis: Stool culture.

ii) Bacteraemia: Occurs usually in infants, old patients, and patients with AIDS. Main features include watery stools followed by septic arthritis, meningitis, etc.
Diagnosis: Stool culture.

iii) Enteric fever (Typhoid):
Incubation period: 1 week.
Main features include fever, headache, myalgia, cough.
Complications may set in like intestinal ulceration, bronchitis, hepatosplenomegaly, etc.
Diagnosis: Culture of stool, urine and blood.

iv) Asymptomatic carrier state

Treatment:

1. Chloramphenicol, or trimethoprim-sulfamethoxazole, or ampicillin.
2. Supportive measures.

5. **Vibrio**

i) **Vibrio parahaemolyticus**

Mode of action:

1. Invasion of intestinal mucosa.
2. Production of toxin.

Sources: Sea food.

Incubation period: 6 hours to 4 days.

Signs and symptoms: Watery diarrhoea, abdominal pain, vomiting.

Diagnosis: Stool culture.

Treatment: Supportive.

ii) **Vibrio cholerae**

Mode of action: Elaboration of an enterotoxin (choleragen).

Sources: All foods, especially sea food like oysters and crabs. In India it is usually a water borne illness.

Incubation period: Few hours to 4 to 5 days.

Signs and symptoms: Explosive watery diarrhoea, with very rapid dehydration and exhaustion. Stools are characteristically rice water in appearance. There is some similarity with acute arsenic poisoning (see page no. 320 for the differentiating points). Metabolic acidosis is usually present.

Diagnosis: Stool culture.

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Treatment:

1. Emergency fluid replacement, preferably Ringer lactate and potassium chloride in severe cases.
2. Tetracycline.

6. **Clostridium botulinum**

It is a spore forming, anaerobic, gram positive bacillus, and produces a food borne illness called **botulism**.

Mode of action: Transmission is through spores which can resist cooking or boiling.[2] These spores germinate at pH above 4.6, and elaborate a neurotoxin which binds to neuromuscular junctions, and inhibits the pre-synaptic release of acetylcholine.

Sources: Canned food. Contaminated food usually smells and tastes bad, but not always.

Incubation period: 6 hours to 1 week.

Signs and symptoms: Mild gastrointestinal symptoms, weakness, sore throat, dry mouth, diplopia, ptosis, dysarthria, and dysphagia, followed by bulbar paralysis, i.e., a bilaterally symmetrical descending motor paralysis. Death usually occurs form respiratory failure.

Table 14.1 Differential Diagnosis of Botulism

Acute intermittent porphyria	Guillain-Barre syndrome
Snake bite	Hysteria
Anticholinergic overdose	Myasthenia gravis
Bulbar polio	Organophosphate poisoning
Tetanus	Tick paralysis
Diphtheria	

Diagnosis:

1. Stool culture.
2. Laboratory analysis of blood, gastric contents, and food.

Rule out myasthenia gravis by performing an **edrophonium challenge test (Tensilon test)**. When 10 mg of edrophonium is given IV, myasthenia will respond dramatically while botulism does not.

Treatment:

1. Decontamination (upto 24 hours post-ingestion).
2. Antitoxin, if available. It must be given early, since it does not make any difference to already paralysed muscles.
3. Guanidine may help as adjunctive therapy. The usual dose is 15 to 30 mg/kg/day.
4. Supportive measures, with special emphasis on ventilatory support.

Usual fatal dose: Few micrograms of botulinum toxin.

7. **Streptococcus**

Infection occurs mainly through contaminated high protein food, and produces a flu-like syndrome without significant gastrointestinal manifestations. Treatment involves administration of penicillin.

8. **Campylobacter**

It is said to be one of the commonest causes of diarrhoea in the world.

Mode of Action:

1. Invasion of intestinal mucosa.
2. Elaboration of enterotoxin.

Sources:

1. Water
2. Milk
3. Meat

Incubation period: 1 to 8 days.

Signs and symptoms: Watery or bloody diarrhoea, along with fever, abdominal pain, and headache. There may be complications like convulsions, bacteraemia, meningoencephalitis, etc.

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Diagnosis:
1. Dark field microscopy of stool.
2. Culture of stool or blood.

Treatment: Mainly supportive measures. In severe cases, erythromycin ethylsuccinate can be given at a dose of 400 mg four times a day. A less satisfying alternative is ampicillin.

9. **Escherichia coli**

Mode of Action:
1. Invasion of intestinal mucosa.
2. Elaboration of enterotoxin.

Sources:
1. Water
2. Meat

Incubation period: 1 to 3 days.

Signs and symptoms: There are 4 forms of the illness (together referred to as Traveller's Diarrhoea).
i. Dysentery-like
ii. Cholera-like
iii. Hemolytic uraemic syndrome
iv. Haemorrhagic colitis

Diagnosis: Stool culture

Treatment:
1. Trimethoprim-sulfamethoxazole
2. Doxycycline

II. **Viral Gastroenteritis**

This is becoming increasingly common today. Main culprits include rotavirus, parvovirus and enteric adenovirus.

Mode of action: Invasion of intestinal epithelial cells with a resultant malabsorption syndrome.

Sources: Water is the main source.

Incubation period: 1 to 3 days.

Signs and symptoms: Vomiting, and watery, foul smelling diarrhoea.

Diagnosis: On the basis of clinical signs, and by a process of exclusion.

Treatment: Supportive measures.

III. **Other Microorganisms**

In addition to bacterial and viral food poisoning, there are illnesses caused by food contamination with microorganisms such as **protozoa** (amoebiasis), and **parasites** (ascariasis, taeniasis, etc.), which are beyond the scope of this book. **Giardia lamblia** is responsible for a significant proportion of diarrhoeal illness in India. The cysts of this protozoon contaminate water, and when this is consumed, newly formed trophozoites invade mucosal walls characterized by a malabsorption syndrome (bloating, abdominal pain, flatus, foul smelling stools, etc). Incubation period is usually 1 to 2 weeks. Treatment involves administration of metronidazole (250 mg 8th hourly for 1 week), or quinacrine hydrochloride (100 mg 8th hourly for 1 week).

Postmortem Appearances (All Forms of Microbial Food Poisoning) :

The outstanding feature in most cases (except botulism) is gastrointestinal congestion. In some cases there is ulceration of intestinal mucosa. Botulism fatalities usually are characterised by a total lack of postmortem findings. Asphyxial signs may be present.

Medicolegal Importance (All Forms of Microbial Food Poisoning):

Cases of mass food poisoning are not uncommon in India, and usually occur during functions or celebrations when food is served to a large number of guests. Contamination may result from unhygienic measures during the preparation or storage of articles of food. Such cases have medicosocial implications rather than any medicolegal significance. The public health authorities must be contacted to take charge of the situation. Medical practitioners coming across cases of food poisoning from public eateries such as hotels and canteens also must report them to the public health officials.

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Isolated cases of food poisoning may have medicolegal importance in rare instances, e.g., the deliberate addition of a culture of microorganisms in the food of an intended victim of homicide.

Botulism represents a special type of problem on account of its distinctive symptomatology and high mortality. Fortunately it is a rare type of food poisoning in India, since canned food (the main source of Clostridium botulinum) is relatively unpopular here. There is no justification for complacency however, on account of the serious nature of the illness.

A common misconception in botulism is that only canned meat products are dangerous and vegetables are safe. In fact more than 60% of the cases of botulism result from consumption of canned vegetable items (tomatoes, beans, mushrooms, etc.). Though it is true that canned food is generally unpopular in India, such items do find a place on shop shelves in uptown areas.

B. *Food Poisoning Through Plants*

I. **Argemone mexicana (Prickly poppy)**

Physical appearance: It is a herbaceous plant belonging to family Papaveraceae, and grows wild in the countryside all over India. It bears spiny leaves, yellow flowers, and oblong, prickly capsules containing a number of small black seeds which resemble mustard seeds. Oil can be extracted from the seeds.

Active principles: Seeds contain berberine and protopine, while the oil contains sanguinarine and dihydrosanguinarine.

Uses: There are no legitimate uses for this plant, but the seeds and oil are used illicitly to adulterate mustard seeds and oil. When such contaminated products are consumed over a period of time, it results in a condition called **epidemic dropsy.**[4]

Signs and symptoms: Epidemic dropsy presents with vomiting, diarrhoea, oedema (especially of the feet; sometimes a generalised anasarca is seen), pleural and pericardial effusions, hepatomegaly, and congestive heart failure with breathlessness. Haemangiomata may occur in the skin and sometimes glaucoma is seen. Hair loss is noted in some cases. Death may result from myocardial damage.

Treatment:

1. Immediate withdrawal of contaminated seeds or oil.
2. Diuretics.
3. Corticosteroids.
4. Symptomatic measures.

Postmortem Appearances:

1. Haemangiomata in the skin.
2. Oedema of feet, or anasarca.
3. Pleural and pericardial effusions.
4. Hepatomegaly.

Medicolegal Importance: Adulteration of mustard oil with argemone oil is common in certain parts of India such as Delhi, Uttar Pradesh, West Bengal, Maharashtra, and Andhra Pradesh. The recent outbreak of epidemic dropsy in Delhi and neighbouring states (July-August 1998), claimed nearly a hundred lives, and has highlighted the catastrophic consequences of edible oil adulteration.

Mushrooms

All mushrooms are not poisonous. In fact the vast majority are not only non-toxic, but are quite delicious to eat. Less than 5% of the thousands of species are poisonous. All toxic mushrooms belong to

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two divisions : *Basidiomycetes* and *Ascomycetes*. Unfortunately, differentiating the poisonous variety from the edible variety is extremely difficult, and only an experienced mycologist can confidently do this. Mushrooms collected from the wild by untrained persons must never be consumed, unless a positive identification is done by an expert. One must never rashly assume that a particular variety of wild mushroom is edible. Such bravado can result in disastrous consequences.

Table 14.2 gives common names of various mushrooms.

Amanita phalloides alone is said to cause more than 50% of all mushroom poisoning, and the *Amanita* genus on the whole accounts for a staggering 95% of fatalities resulting from mushroom toxicity. Therefore, the following discussion will centre around this particular species.

Table 14.2 Common names of some toxic mushrooms

Botanical name	Common name
Amanita muscaria	Fly agaric
Amanita pantherina	Panther cap or False blusher
Amanita phalloides	Death cap
Amanita virosa	Destroying angel
Clitocybe dealbata	Sweater
Coprinus atramentarius	Inky cap
Gyromitra esculenta	False morel
Psilocybe caerulipes	Blue foot
Psilocybe semilanceata	Liberty cap

Physical appearance: The mature *Amanita phalloides* has an olive green cap, white gills, a ring around the stalk, a cup or volva at the bottom of the stalk and white spores. Similar features are seen in other *Amanita* species with only minor differences.

Active principles: Phalloidins (only in Amanita species), Amatoxins (most important toxic principle) and Virotoxins.

Mode of action: Amatoxins are powerful inhibitors of cellular protein synthesis. Cells with high replication rates (liver, kidney, intestinal cells) develop necrosis. Liver usually shows centrilobular necrosis.[5]

Signs and symptoms: There is usually an incubation period of 6 to 12 hours.[6] The signs and symptoms fall into the following three stages :

1. Gastroenteritis stage: lasts for 24 hours, and is characterised by cholera-like diarrhoea, abdominal pain, vomiting, fever, etc.
2. Latent stage: lasts for a variable period during which most of the symptoms subside, and the attending physician is liable to come to the erroneous conclusion that his patient had just suffered an attack of gastroenteritis, of which he has been cured. The patient is therefore usually discharged, but comes back in 2 to 3 days in a moribund condition.
3. Hepatorenal stage: jaundice, delirium, confusion, metabolic acidosis, coagulopathies, renal failure, and coma. Death occurs in 20 to 40% of the cases.

Diagnosis:

Meixner Test: It detects amatoxins in stools or gastric samples (or in the mushroom specimen itself). Since amatoxins are present in all toxic mushrooms, the test can be done for diagnosis of all types of mushroom poisoning.

Procedure

i) Dilute the stool or gastric sample with methanol, centrifuge, and filter it. Add a drop or two to a piece of newspaper. If the test is to determine amatoxins in a given mushroom, squeeze a drop or two of juice from it on to a piece of pulp paper (e.g., newspaper).

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ii) Encircle the spot with a pencil and dry it.
iii) Add a few drops of conc. HCl to the dry spot. If a blue colour forms within 15 to 20 minutes, it is indicative of the presence of amatoxins.

Treatment:

1. Gut decontamination in the usual way.
2. Correction of dehydration and electrolyte imbalance.
3. Forced diuresis. Hemoperfusion in severe cases
4. A number of drugs have been claimed as antidotes including Penicillin G, thioctic acid, insulin, human growth hormone, vitamin C, etc. The actual efficacy of any of these drugs is questionable.
5. Supportive measures, with special reference to treatment of hepatic failure.

Usual fatal dose:2 to 3 mushrooms, (A.Phalloides).

While there are a number of other causes of food poisoning besides those discussed in this chapter, such as poisonous fish (**icthyotoxicosis**), chemicals, food additives (e.g., monosodium glutamate which causes the so-called *Chinese restaurant syndrome*), etc., they are beyond the scope of this handbook, and the reader is advised to consult standard works on toxicology for the same.

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Annexures

ANNEXURE - 1 OATHS AND CODES OF ETHICS FOR MEDICAL PROFESSIONALS

HIPPOCRATIC OATH

I will look upon him who shall have taught me this art even as one of my parents. I will share my substance with him and I will supply his necessities, if he be in need. I will regard his offspring even as my own brethren and I will teach them this art, if they would learn it, without fee or covenant. I will impart this art by precept, by lecture, and by every mode of teaching, not only to my own sons, but to the sons of him who has taught me, and disciples bound by covenant and oath, according to the Law of Medicine.

The regimen I adopt, shall be for the benefit of my patient according to my ability and judgement, and not for their hurt or for any wrong. I will give no deadly drug to any though it be asked of me, nor will I counsel such, and especially I will not aid a woman to procure abortion. Whatsoever house I enter, there will I go for the benefit of the sick, refraining from all wrong doing or corruption, and especially from any act of seduction, of male or female, of bond or free. Whatsoever things I see or hear concerning the life of men, in my attendance on the sick or even apart therefrom which ought not be noised abroad, I will keep silence thereon, counting such things as sacred secrets.

The general principles mentioned in the HIPPOCRATIC OATH have been updated by the World Medical Association. The modernised versions of the Hippocratic Oath are the Declaration of Geneva, as adopted by the Third General Assembly of World Medical Association at Geneva, Switzerland, in September 1948, and the International Code of Medical Ethics, as adopted by the General Assembly of the World Medical Association held in London, in October, 1948.

THE DECLARATION OF GENEVA (Sept. 1948; Amended Aug. 1968)

At the time of being admitted as a member of the Medical Profession:

I solemnly pledge myself to consecrate my life to the service of humanity,
I will give to my teachers the respect and gratitude which is their due,
I will practice my profession with conscience and dignity,
The health of my patient will be my first consideration,

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I will respect the secrets which are confided in me, even after the patient has died,
I will maintain by all the means in my power, the honour and the noble traditions of the medical profession,
My colleagues will be my brothers,
I will not permit considerations of religion, nationality, race, party politics or social standing to intervene between my duty and my patients,
I will maintain the utmost respect for human life from the time of conception,
Even under threat, I will not use my medical knowledge contrary to the laws of humanity,
I make these promises solemnly, freely, and upon my honour.

THE INTERNATIONAL CODE OF MEDICAL ETHICS (Oct. 1949)

Duties of Doctors in General:

- A doctor must practice his profession uninfluenced by motives of profit. The following practices are deemed unethical:
  a. Any self-advertisement, except such as is expressly authorised by the code of medical ethics.
  b. Collaboration in any form of medical service in which the doctor does not have professional independence.
  c. Receiving any money concerning the services rendered to a patient other than a proper professional fee, even with the knowledge of the patient.
  d. Any act or advice, which could weaken physical or mental resistance of a human being.
- A doctor is advised to use great caution in divulging discoveries or new techniques of treatment.
- A doctor should certify or testify only to that which he has personally verified.

Duties of the Doctor to the Sick:

- A doctor must always bear in mind the obligation of preserving human life.
- A doctor owes to his patient complete loyalty and all the resources of his science. Whenever an examination or treatment is beyond his capacity, he should summon another doctor who has the necessary ability.
- A doctor shall preserve absolute secrecy on all he knows about his patients because of the confidence entrusted in him.
- A doctor must give emergency care and render humanitarian duty at any time, unless he is assured that others are willing and able to give such care.

Duties of Doctors to Each Other:

- A doctor ought to behave with his colleagues as he would have them behave towards him.
- A doctor must not entice patients from his colleagues.
- A doctor must observe the principles of the 'The Declaration of Geneva' approved by the World Medical Association.

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Discrimination in Medicine:

The Declaration of Geneva, states inter alia, that:

'I (as a medical practitioner) will not permit considerations of religion, nationality, race, party politics, or social standing to intervene between my duty and my patient.'

The 27th meeting of the World Medical Association (WMA) held in Munich in 1973 resolved therefore, that the

'WMA vehemently condemns colour, political, and religious discrimination of any form in the training of medical practitioners and in the practice of medicine and in the provision of health services for the peoples of the world.'

DOCTORS AND THE MEDIA

Increasing public interest in health matters has brought doctors more and more into contact with the media. Registered medical practitioners should be aware of the ethics involved in dealing with the media. The British Medical Association in its *Handbook of Medical Ethics* 1981, suggests the following guidelines:

- Those doctors able to comment authoritatively on medical subjects should be prepared to do so in order that the public may be informed. Those doctors able to help the public with information, should regard talking to the media as an extension of their medical practice.
- A doctor has the responsibility to ensure that when a subject under discussion is controversial with regard to the medical profession, the producer or the editor of a programme is made aware of that fact.
- It is unacceptable for a doctor to publicly discuss his own ability in a particular field in such a way as to imply that his methods are superior to those of other doctors.
- It is acceptable for a doctor's identity to be revealed in the following circumstances:
  - When it does not accentuate his professional stature,
  - When it is in the public interest,
  - When he is speaking on behalf of an identifiable section of the profession,
  - When using media primarily aimed at doctors.
- A doctor may use his own name in connection with subjects other than medicine. When discussing a medical subject, he may be named only if he confines himself to general terms avoiding discussion of individual cases.
- In the field of general health education, the name and the relevant qualifications of the doctor may be given to enhance credibility.
- Doctors making statements on behalf of known organisations may be named when this is in the public interest. However, a doctor must not exploit the media to promote any organisation in which he has a financial interest.
- No doctor should enter into a correspondence with an individual member of the lay pubic as a result of any publicity gained by the doctor.

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ANNEXURE - 2 METHODS OF ASSESSING THE DEPTH OF TOXIC COMA

Table 1 GLASGOW COMA SCALE

Feature observed	Score
1. *Eye Opening*	
a. Spontaneous	E4
b. To speech	3
c. To pain	2
d. Nil	1
2. *Best Motor Response*	
a. Obeys	M6
b. Localises	5
c. Flexes (withdrawal)	4
d. Flexes abnormally (decorticate rigidity)	3
e. Extends (decerebrate rigidity)	2
f. Nil	1
3. *Best Verbal Response*	
a. Oriented	V5
b. Confused conversation	4
c. Inappropriate words	3
d. Incomprehensible sounds	2
e. Nil	1

Table 2 REED'S CLASSIFICATION OF COMATOSE PATIENTS

Group 0	Arousable
Group 1	Respond to painful stimuli and have intact reflexes
Group 2	Do not respond to painful stimuli - most reflexes are normal
Group 3	Do not respond to painful stimuli - most reflexes are absent
Group 4	Deeply comatose, with respiratory and/or circulatory failure

* *An example of a minimally painful stimulus is to pinch the cheek lightly, while a suitable maximally painful stimulus is to rub the patient's sternum with the knuckles of the clenched fist.*

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Table 3 CNS INTOXICATION SCALE

Depressants		Stimulants	
Grade	**Features**	**Grade**	**Features**
0	Asleep, but can be aroused	-	-
1	Semicomatose, withdraws from painful stimuli, reflexes intact	1.	Restlessness, irritability, insomnia, tremor, hyperreflexia sweating, mydriasis
2.	Comatose, does not withdraw from painful stimuli, reflexes intact	2.	Confusion, hypertension, tachypnoea, tachycardia, extrasystoles
3.	Comatose, most reflexes lost, no depression of CVS or respiration	3.	Delirium, mania, arrhythmia, hyperpyrexia
4.	Comatose, reflexes absent, respiratory and/or circulatory failure.	4.	Convulsions, coma, and circulatory collapse

Adapted from **Pillay VV.** *Toxic coma: Problems of diagnosis and assessment.* J Indian Acad Forensic Med **2001;** 22 : 12-15.

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ANNEXURE - 3 GOALS AND OBJECTIVES IN FORENSIC MEDICINE AND TOXICOLOGY

(as per the Medical Council of India Guidelines, 1997)

GOALS

The broad goal of the teaching of undergraduate students in Forensic Medicine is to produce a physician who is well informed. He/She should also be capable of making observations and inferring conclusions by logical deductions, to set enquiries on the right track in criminal matters and connected medicolegal problems. He/She should acquire knowledge of law in relation to medical practice, medical negligence, and respect for codes of medical ethics.

OBJECTIVES

1. KNOWLEDGE

At the end of the course, the student should be able to:

- identify the basic medicolegal aspects of hospital and general practice,
- define the medicolegal responsibilities of a general physician while rendering community service either in a rural primary health centre or an urban health centre,
- appreciate the physician's responsibilities in criminal matters and respect for the codes of medical ethics,
- diagnose, manage, and identify the legal aspects of common acute and chronic poisonings,
- describe the medicolegal aspects and findings of postmortem examination in case of death due to common unnatural conditions and poisonings,
- detect occupational and environmental poisoning, devise measures of prevention of common poisoning, and deal with legal aspects, particularly pertaining to Workmen's Compensation Act,
- describe the general principles of analytical toxicology.

2. SKILLS

At the end of the course, the student should be able to:

- make observations and logical inferences in order to initiate enquiries in criminal matters and medicolegal problems,
- diagnose and treat common emergencies in poisoning and manage chronic toxicity,
- make observations and interpret findings at postmortem examination,
- observe the principles of medical ethics in the practice of his profession.

3. INTEGRATION

The department of Forensic Medicine & Toxicology shall provide an integrated approach towards allied disciplines like Pathology, Radiology, Forensic Sciences, Hospital Administration, etc., to impart training regarding medicolegal responsibilities of physicians at all levels of health care. Integration with relevant disciplines will provide scientific basis of clinical toxicology e.g., medicine, pharmacology, etc.

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ANNEXURE - 4 MODEL SCHEME OF LECTURES IN FORENSIC MEDICINE AND TOXICOLOGY

(as per the Medical Council of India (MCI) Recommendations 1997)

II MBBS : III, IV & V Semesters. Total Classes : 70 (1 hour each)

INTRODUCTION & JURISPRUDENCE

Classes

I. INTRODUCTION TO FORENSIC MEDICINE — 1
1. Definition, Synonyms, Historical Aspects, Modern Forensic Medicine - Sub divisions, etc.

II. LEGAL PROCEDURE — 4
1. Introduction, Important Legal Terms (Glossary), Courts, Inquests.
2. Recording of Evidence in Court, Medical Evidence, Dying Declaration.
3. Witness, Doctor in Witness Box, Exhumation.
4. Workmen's Compensation Act (Principles).

III. MEDICAL ETHICS — 6
1. Introduction, M.C.I & S.M.C : Functions.
2. Registered Medical Practitioner : Duties & Privileges.
3. Infamous Conduct (Professional Misconduct), Punishments, Appeal.
4. Malpractice (Negligence).
5. Consumer Protection Act (Principles), Medical Idemnity Insurance.
6. Consent : in Medical and Medicolegal Practice.
7. Euthanasia.

FORENSIC PATHOLOGY

IV. FORENSIC IDENTITY — 2
1. Introduction : Definition, Types, Corpus delicti, Factors Establishing Identity, Forensic Odontology (Basics), DNA Finger Printing (Principles).
2. Determination of Age & Sex in Living and Dead Individuals.
3. Medicolegal Importance of Age

V. THANATOLOGY — 5
1. Introduction : Types of Death : Somatic, Molecular, & Brain Stem, Organ Transplantation.
2. Sudden Death.

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3. PM Changes : Immediate.
4. PM Changes : Early.
5. PM Changes : Late, Adipocere, Mummification.
6. Medicolegal Importance of Death

VI. MEDICOLEGAL AUTOPSY 2

1. Objectives, Rules, Routine Procedures, Negative Autopsy.
2. Skeletal Remains Examination.

VII. ASPHYXIAL DEATHS 5

1. Introduction, Pathophysiology, General Signs & Symptoms of Asphyxia.
2. Hanging : Definition, Types, Causes of death, PM Findings, MLI.
3. Strangulation, Suffocation, Traumatic Asphyxia : Definition, Types, Causes of Death, PM Findings, MLI.
4. Drowning : Definition, Types, Mechanisms, Causes of Death, Medicolegal Aspects.
5. Drowning : PM Findings, Diatom Test.

CLINICAL FORENSIC MEDICINE

VIII. TRAUMA (MECHANICAL INJURIES) 10

1. Introduction : Definition (Injury & Hurt), Classification (Clinical).
2. Blunt Force Trauma : Abrasions, Contusions, Lacerations (including complications and MLI).
3. Sharp Force Trauma : Incised, Stab and Chop wounds (including complications & MLI).
4. Firearms, Firearm Injuries, P.M. Examination of Firearm Injury Death.
5. Head Injury : Mechanisms, Injury to Scalp, Skull, Brain, Intracranial Haemorrhages.
6. Spinal Injuries.
7. Road Traffic Accidents, Medicolegal Aspects of Trauma.
8. Injury due to Burns & Scalds, Injury due to Electricity, Lightning etc.

IX. SEXUAL JURISPRUDENCE 3

1. Explanation & MLI of Virginity, Pregnancy, MTP, Criminal Abortion, Delivery, Paternity, Impotence, Sterility, Incest, Rape, Sodomy, Paraphilias.
2. Examination of Rape Case (in detail).
3. Examination of Sodomy Case (in detail).

X. INFANTICIDE 2

1. Definition, Explanation of Viability, Haase's Rule, Live Birth, Dead Birth, Still Birth, Tests of Live Birth, Time of Survival, Cause of Death.
2. Battered Baby Syndrome & Sudden Infant Death Syndrome

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XI. FORENSIC PSYCHIATRY 2

1. Explanation of Common Psychiatric Terms, Definition and classification of Insanity, Feigned Insanity, McNaughten's Rule.
2. Civil & Criminal Responsibilities of the Insane, Principles of Mental Health Act 1987, Restraint of the Insane.

TOXICOLOGY

XI. GENERAL PRINCIPLES 5

1. Definitions, Epidemiology of Poisoning.
2. M.L. Aspects of Poisoning, Laws on Poisons - Schedules, IPC Sections (relevant), Duties of Doctors in Suspected Poisoning.
3. Diagnosis & Management of Poisoning (General Measures).
4. Preservation & Despatch of Viscera to FSL
5. Analytical Toxicology (Principles & Basics : Bedside & Common Lab Tests, TLC, HPLC, GC, etc).
6. Occupational & Environmental Toxicology.
7. Classification of Poisons.

XII. CORROSIVES (CAUSTICS) 2

1. Inorganic : Sulphuric, Nitric and Hydrochloric Acid.
2. Organic : Phenol, Oxalic Acid, Formic Acid.

XIII. IRRITANTS 6

1. Inorganic Non-Metallic - Phosphorus, Halogens.
2. Inorganic Metallic - Arsenic, Lead, Mercury, Copper, Iron, thallium
3. Organic (Plant) : Abrus, Castor, Croton, Calotropis, Capsicum, Semecarpus, Ergot.
4. Organic (Animal) : Snake Bite, Insect sting (Bee, Wasp), Envenomation due to Centipede, Scorpion, etc.
5. Mechanical Irritants.

XIV. INEBRIANTS 2

1. Ethanol
2. Methanol
3. Ethylene Glycol & Isopropanol
4. Benzodiazepines

XV. SOMNIFEROUS DRUGS, SEDATIVE-HYPNOTICS, & SPINAL POISONS 2

1. Opium & Derivatives
2. Barbiturates
3. Strychnine

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XVI. DELIRIANTS 2

1. Datura, Cannabis, Cocaine.

XVII. PESTICIDES 2

1. Organophosphates, Carbamates, Organochlorines.
2. Pyrethroids, Aluminium Phosphide, etc.

XVIII. CARDIOTOXIC POISONS 1

1. Digitalis, Oleander, Aconite, Nicotine.

XIX. ASPHYXIANTS 2

1. Carbon Monoxide, Cyanide

XX. HOUSEHOLD POISONS 1

1. Kerosene, Cleaning Agents, Disinfectants, Cosmetics, etc.

XXI. THERAPEUTIC DRUGS 1

1. Salicylates, Paracetamol, Antidepressants, Antipsychotics, Insulin

XXII. FOOD POISONING 1

1. Bacterial, Viral, Mushrooms, etc.

XXIII. DRUG ABUSE 1

1. Alcohol, Tobacco, Narcotics, Hallucinogens, Stimulants, etc.

Distribution of Lectures (Hours)

Forensic Medicine	:	42
Toxicology	:	28
Total		70

Distribution of Marks (100)

Forensic Medicine	:	70
Toxicology	:	30
Total		100

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ANNEXURE - 5 MODEL SCHEME OF PRACTICAL EXERCISES IN FORENSIC MEDICINE

Total Classes : 30 (1 hour each)

1. **EXERCISE I : Autopsy Case**
   Analysis of postmortem certificate -
   - An extract of a postmortem certificate is given, with 5 to 10 questions.
   - Common cases are dealt with - Trauma, asphyxia, poisoning, etc.

2. **EXERCISE II : Certificate Case**
   - Wound certification
   - Drunkenness certification
   - Death certification
   - Potency certification
   - Viscera analysis
   - Clinical toxicology case

3. **EXERCISE III : Medico-legal Problem**
   Discussion case involving the elucidation of common medico-legal problems relating to forensic pathology, clinical forensic medicine, or toxicology. An imaginary scenario is presented, and the student is expected to discuss and provide solutions or answers.

Note:

- Practical Record Book evaluation should be part of day to day assessment.
- Exercises such as X-ray analysis for age estimation, examination of skeletal remains, etc., should rightfully be part of P.G. training in Forensic Medicine, and must preferably be excluded from the undergraduate course.
- The utility of having "spotters" in the practical examination is of dubious value. They are generally considered to be obsolete in relation to practical evaluation, by Medical Educationists.

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ANNEXURE - 6 SUGGESTED FURTHER READING IN FORENSIC MEDICINE & TOXICOLOGY

FORENSIC MEDICINE

1. *Parikh's Textbook of Medical Jurisprudence, Forensic Medicine and Toxicology*
C K Parikh
CBS Medical Publishers & Distributors, New Delhi.

2. *HWV Cox's Medical Jurisprudence and Toxicology*
B. Knight, V B Sahai, U S Sinha, and M L Singhal
The Law Book Company Pvt. Ltd., Allahabad.

3. *Modi's Medical Jurisprudence and Toxicology*
B V Subrahmanyam
Butterworths India, New Delhi.

4. *Handbook of Forensic Pathology*
V J M Di Maio and S E Dana
Viva Books Pvt. Ltd., New Delhi.

TOXICOLOGY

1. *Modern Medical Toxicology*
V V Pillay
Jaypee Brothers Medical Publishers, New Delhi.

2. *Diagnosis and Management of Common Poisoning*
P Aggarwal and J P Wali
Oxford University Press, New Delhi.

3. *Acute Poisoning : Diagnosis and Managment*
A T Proudfoot
Butterworth - Heinemann Ltd, Oxford, UK.

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